CPR, Cooling and Airway

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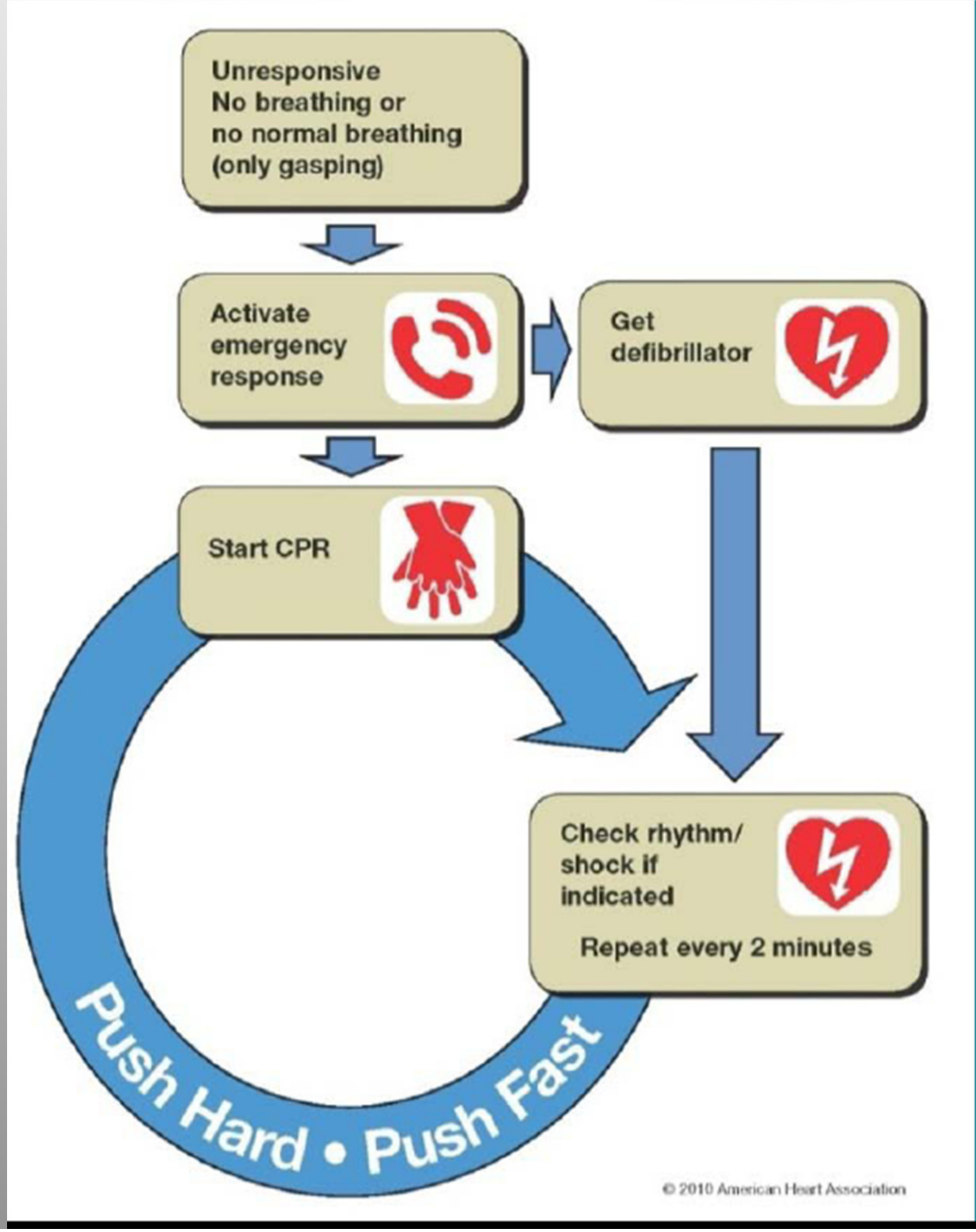
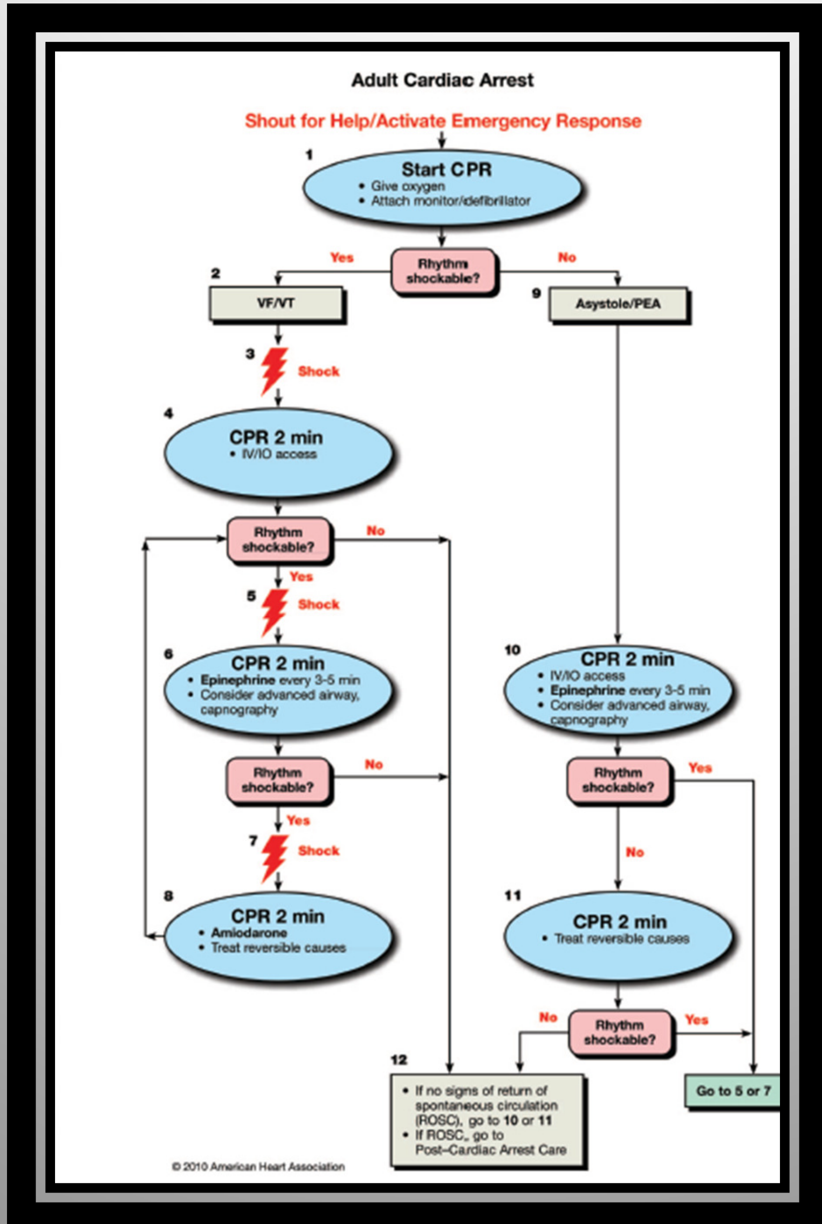
Out of Hospital Cardiac Arrest OHCA

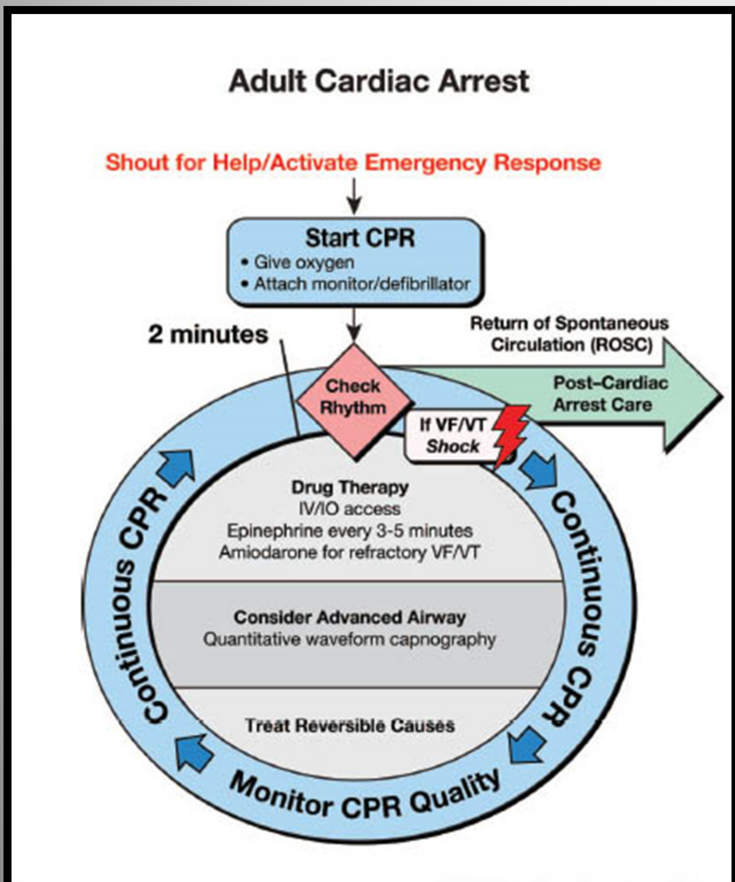
- 350,000 – 450,000 deaths/year in U.S.
- 90% in patients without identified risk factors
- Majority due to Coronary Artery Disease (CAD)
- OHCA is first manifestation of CAD in >50%
- 500-600 OHCA/year Indianapolis


Unresponsive
No breathing or
no normal breathing
(only gasping)
Activate
emergency
response
Get
defibrillator
Start CPR
Check rhythm/
shock if
indicated
Repeat every 2 minutes
Push Hard • Push Fast
© 2010 American Heart Association

2010 AHA CPR Guidelines

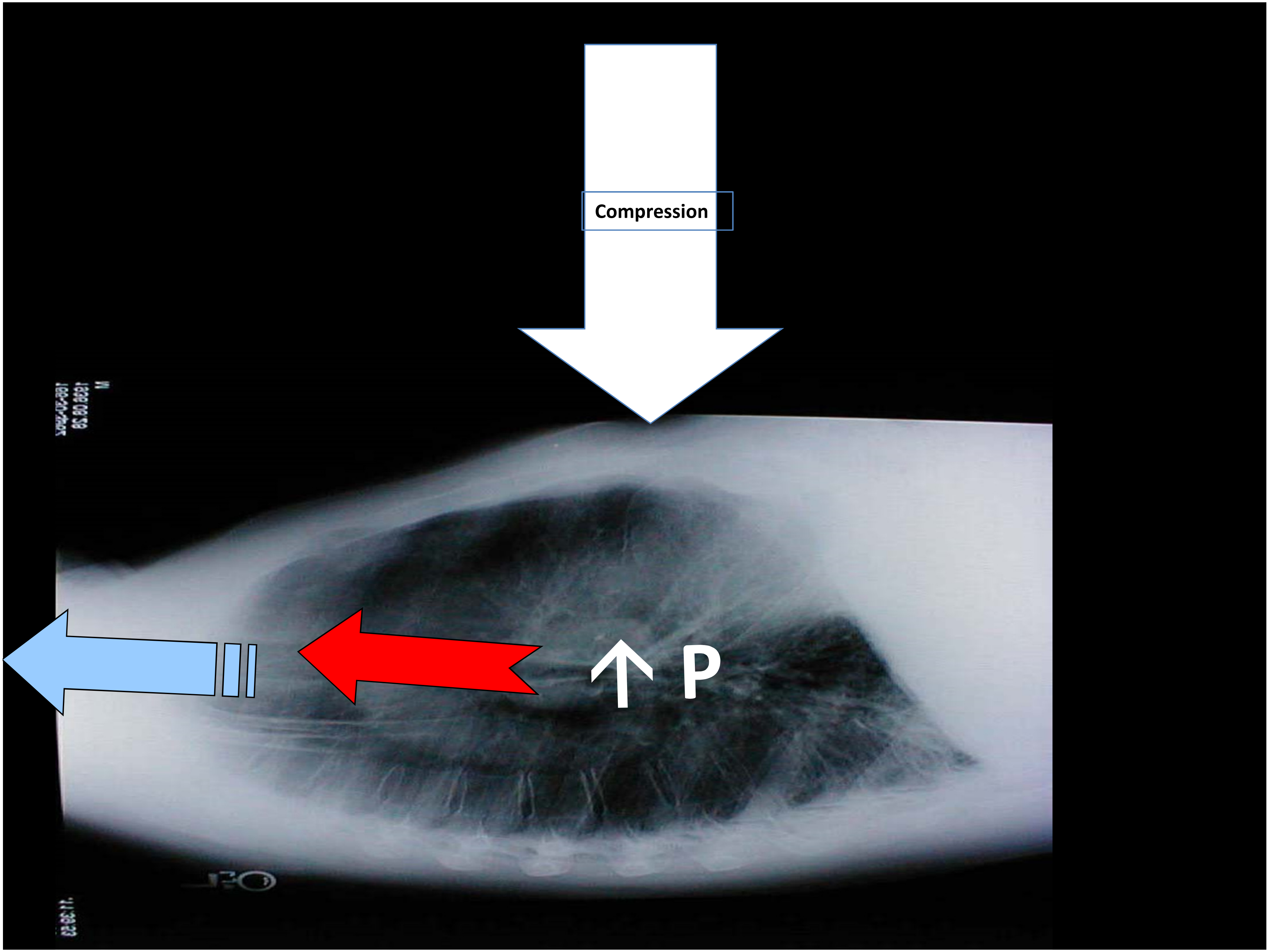


Emphasis on Compressions



- $\geq$ 2 inches (5 cm)
- Allow complete recoil of chest wall
- $\geq$ 100/minute
- Minimal Interruptions
- Avoid excessive ventilations
- Quantitative wave form capnography
  - If $ETCO_2$ < 10 mmHg attempt to improve CPR quality

Physiology of CPR


Compression
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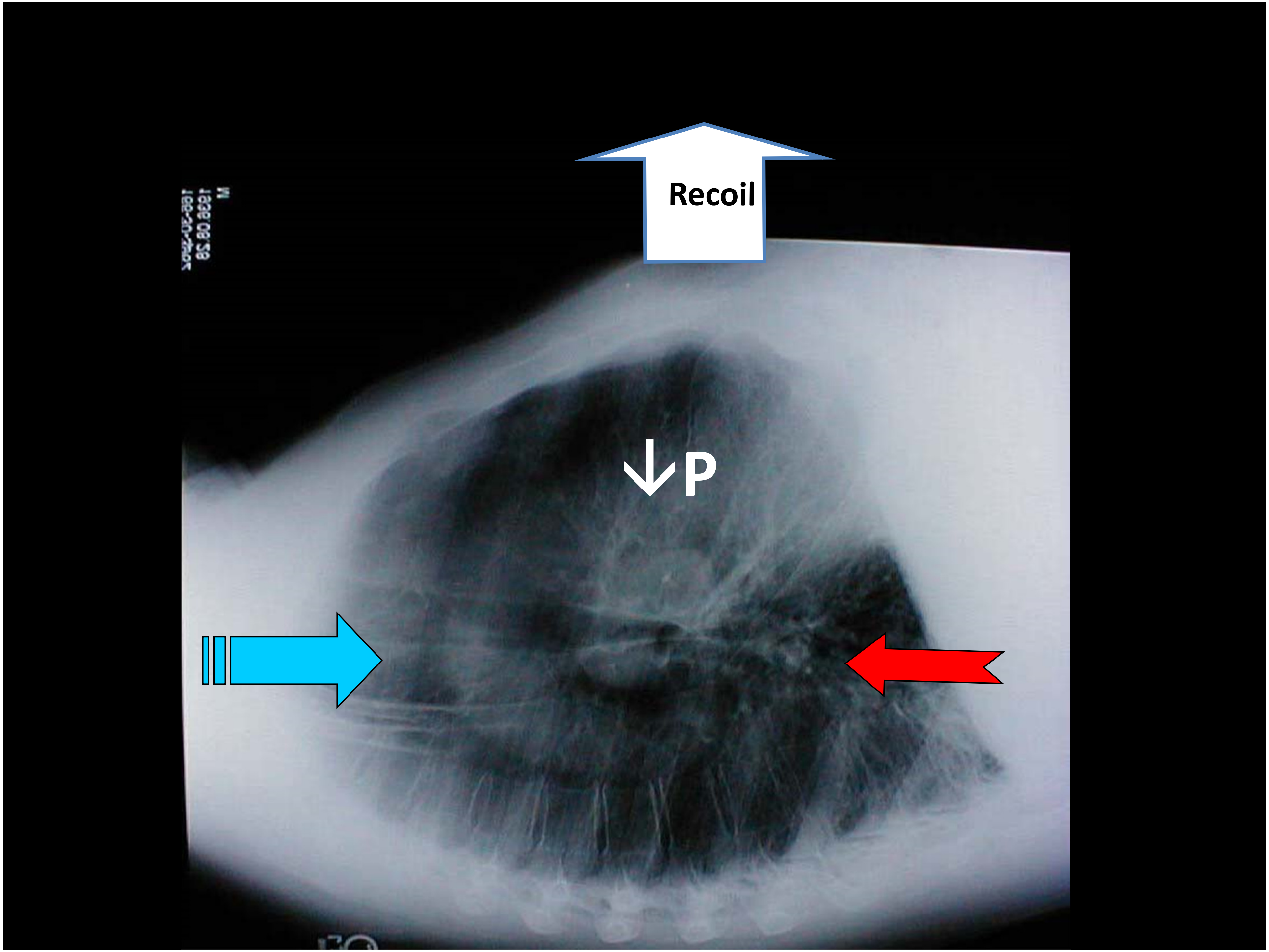
Recoil
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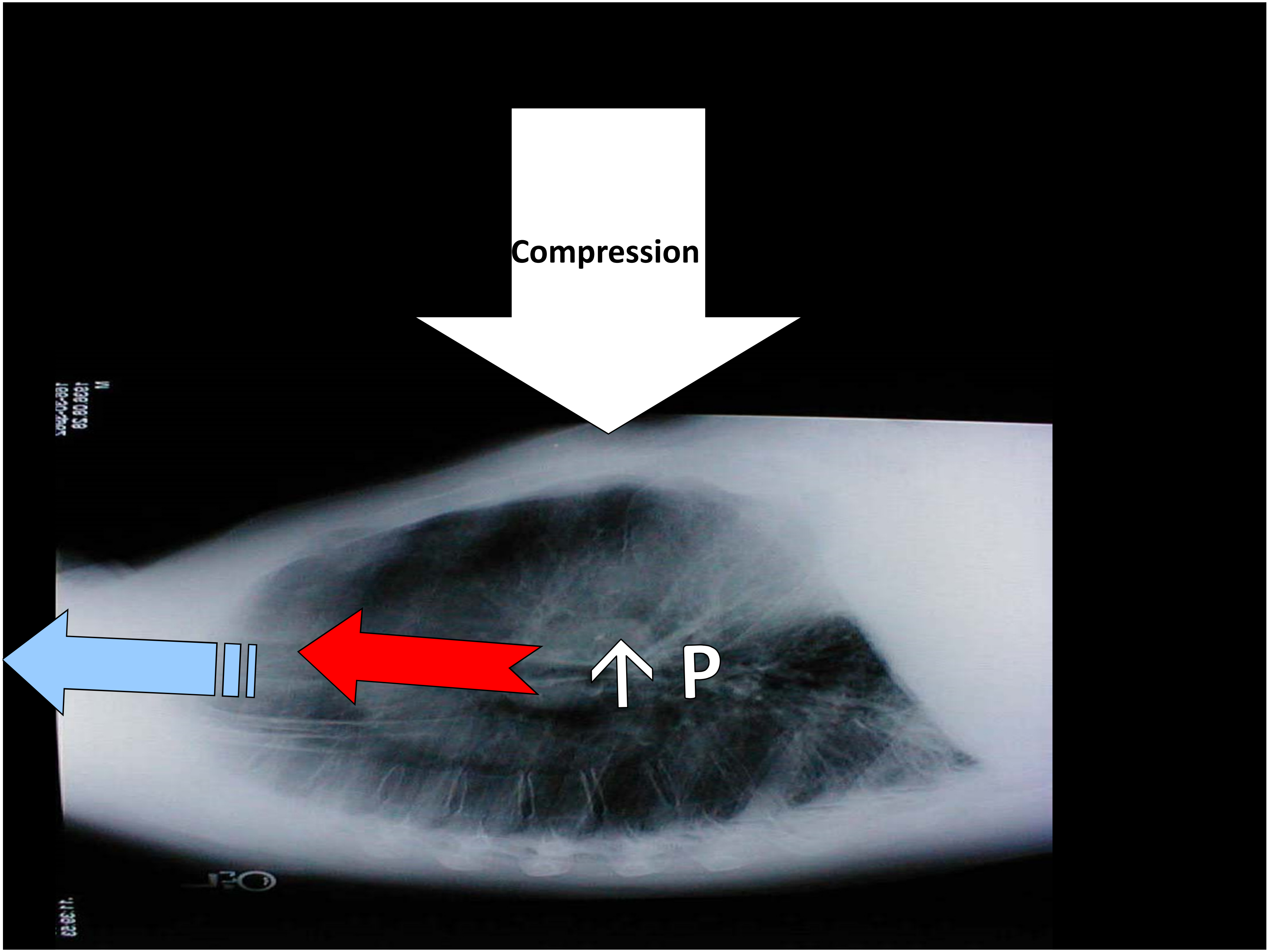
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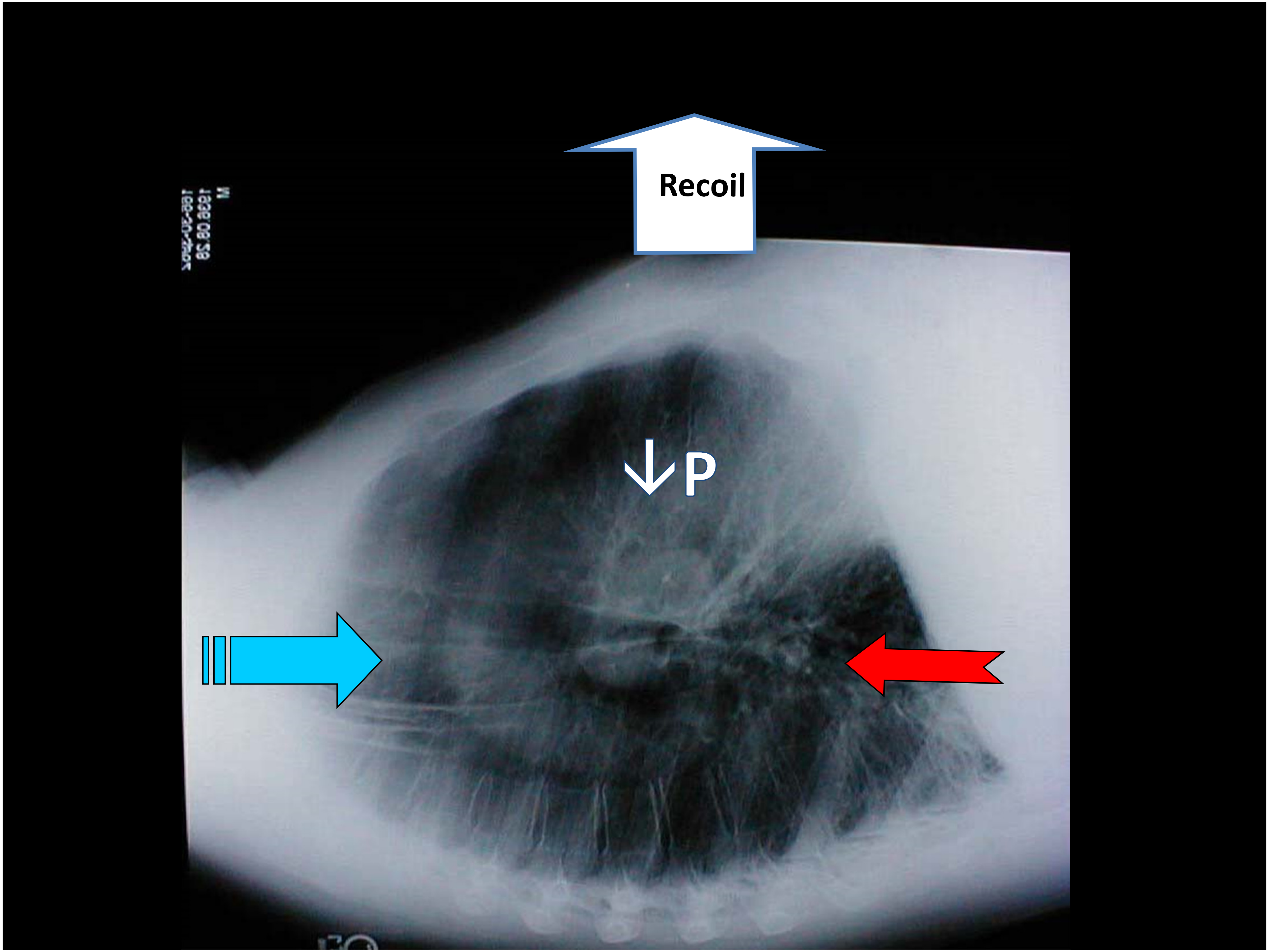
Recoil
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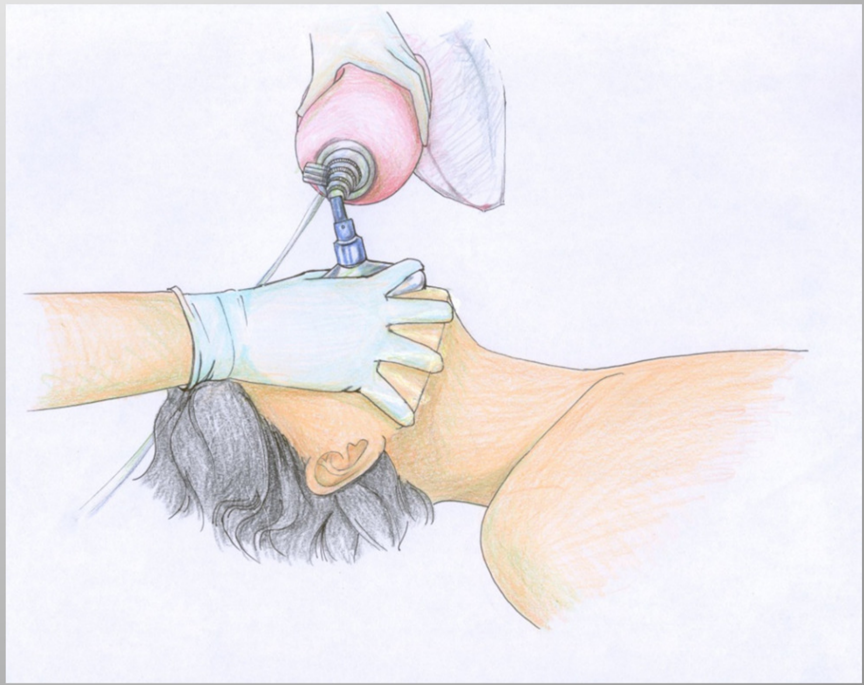
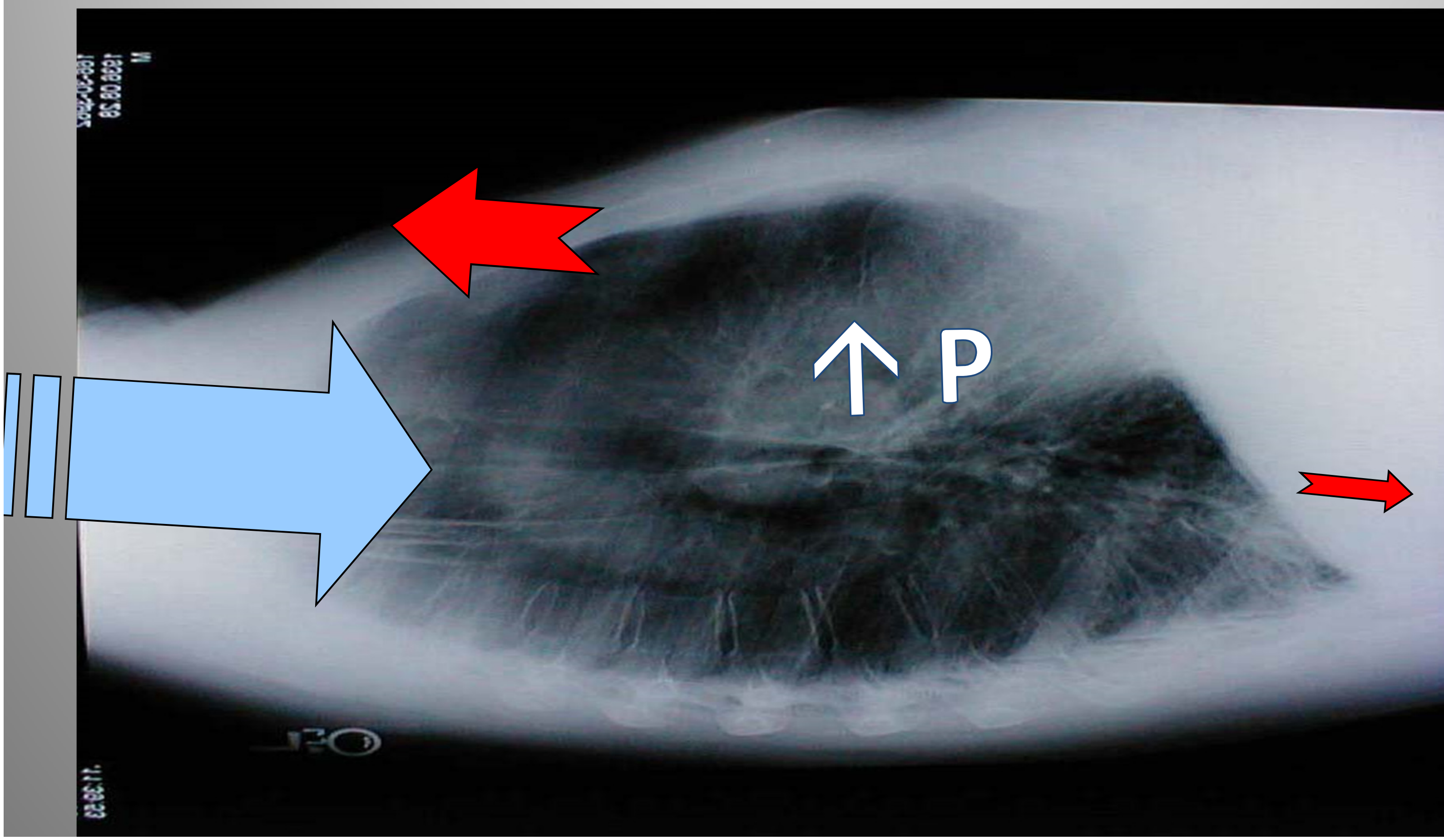
Avoid Excessive Ventilations

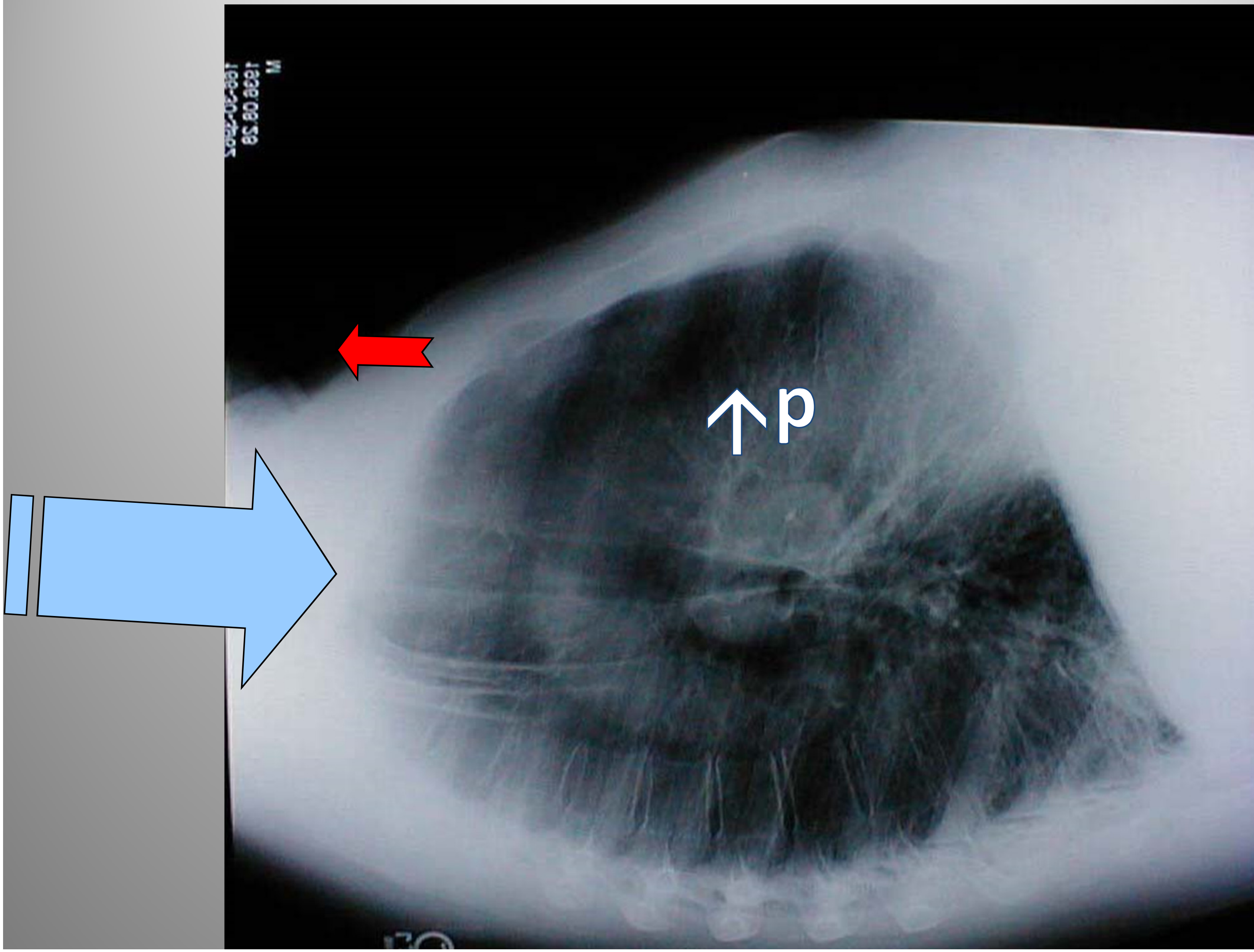
Positive Pressure Ventilation



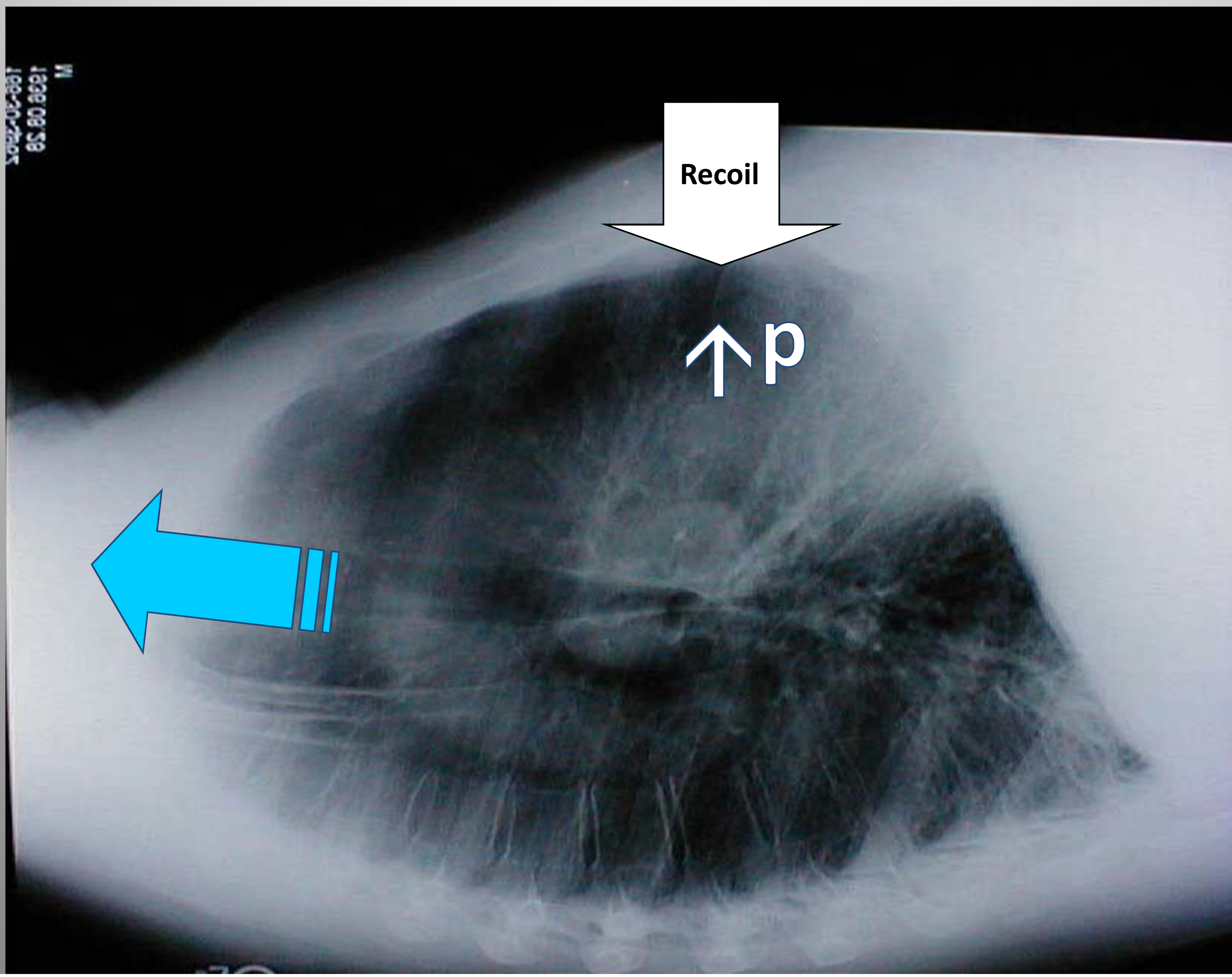
Positive Pressure Ventilation



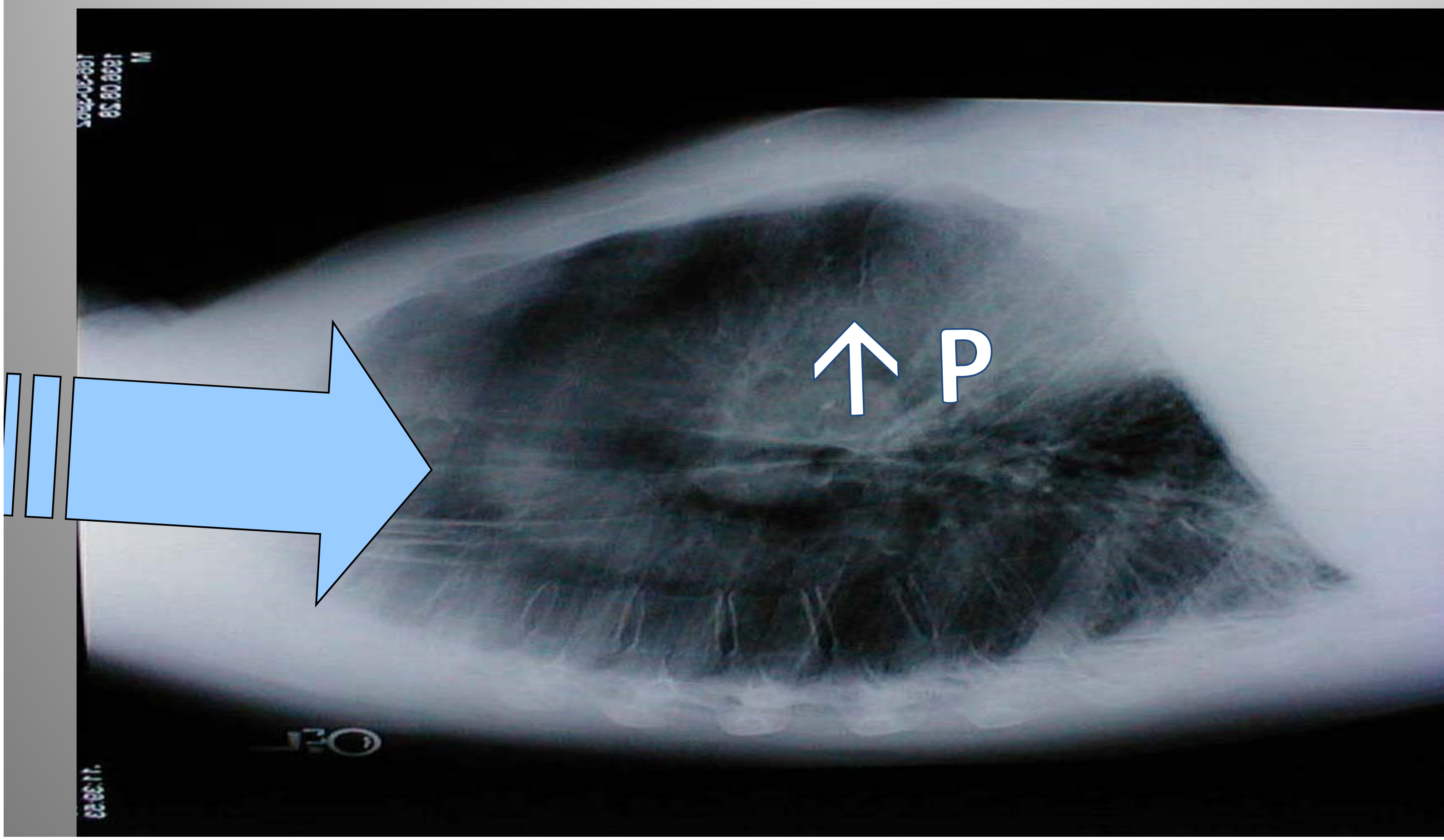
Positive Pressure Ventilation



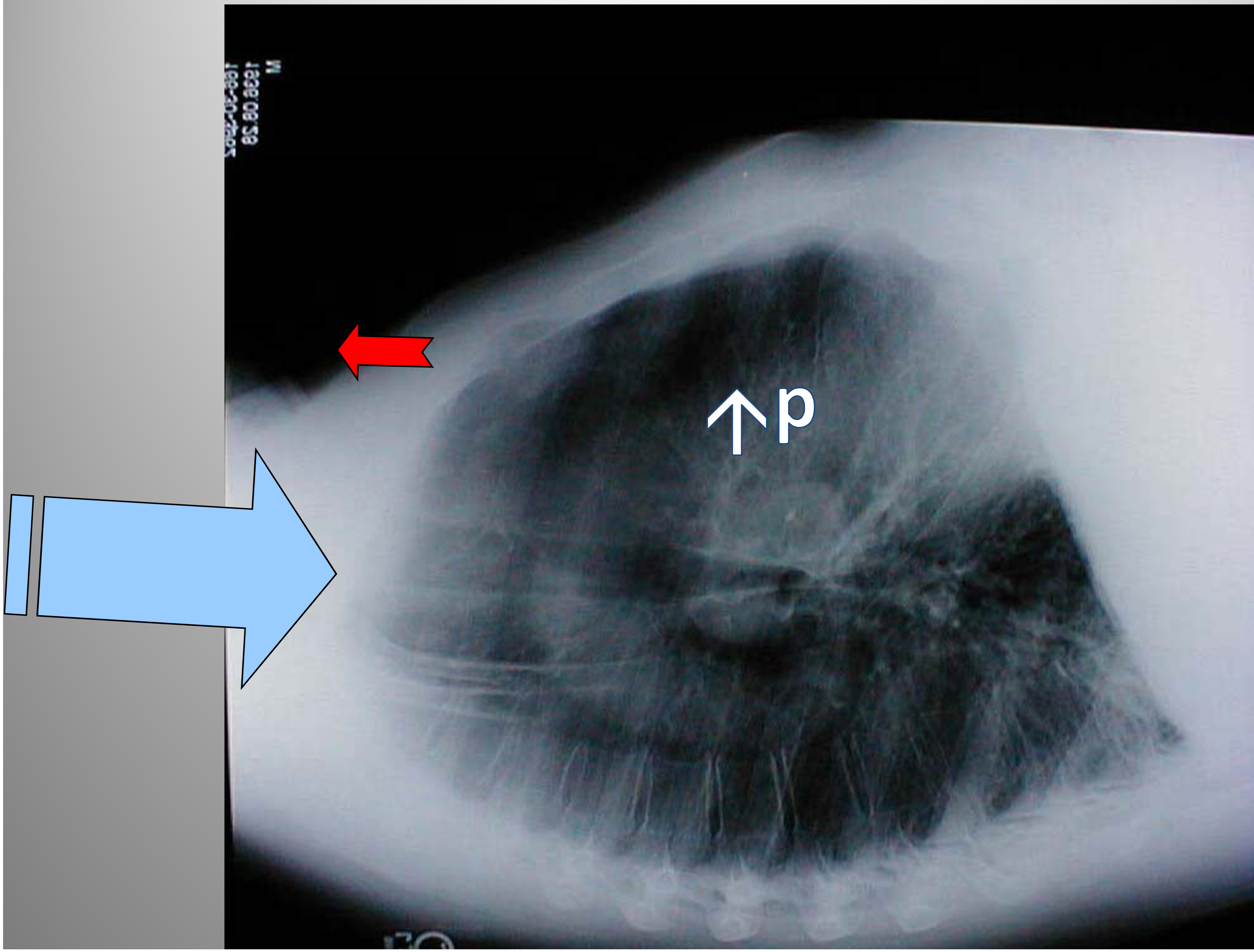
Passive Exhalation



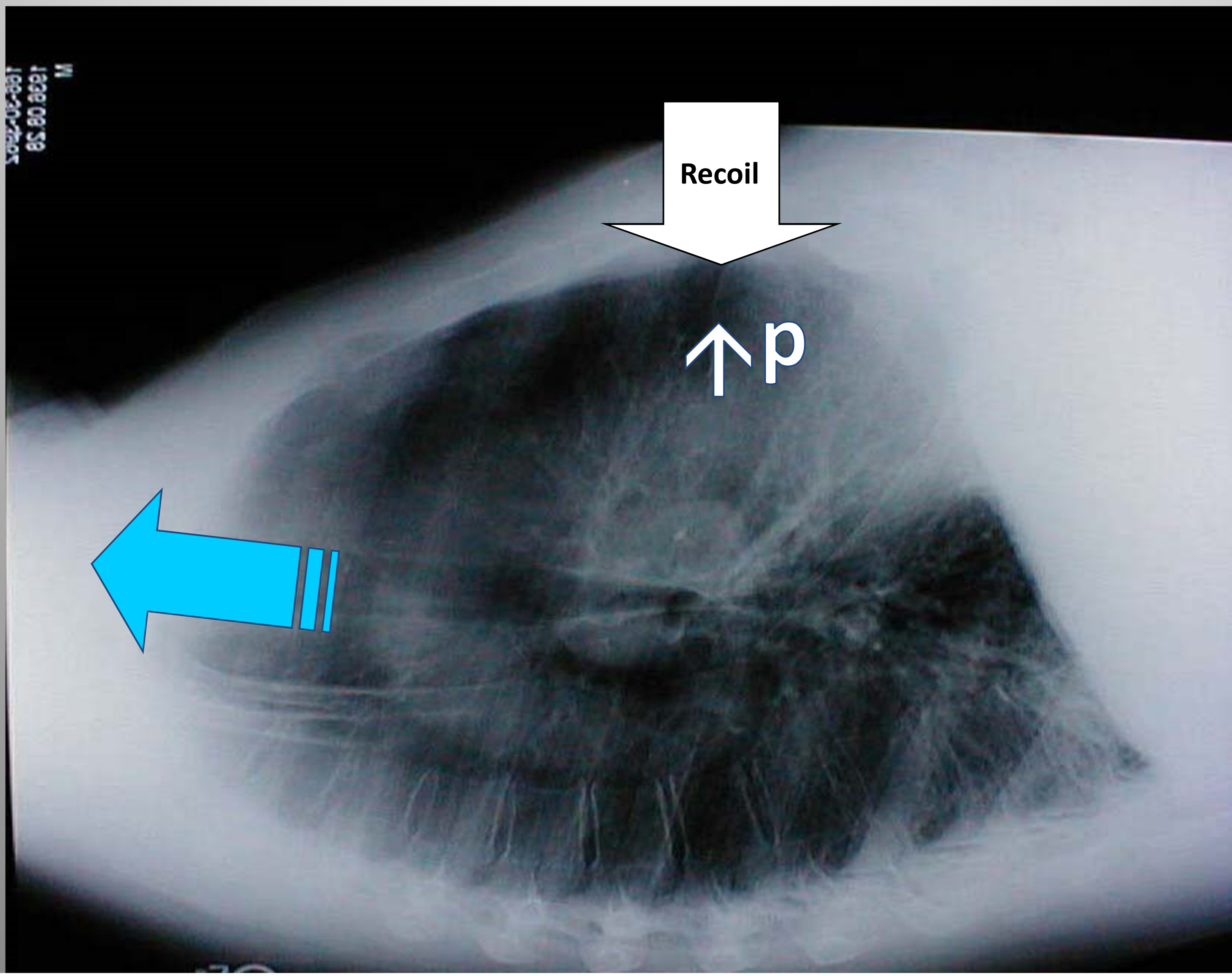
Positive Pressure Ventilation



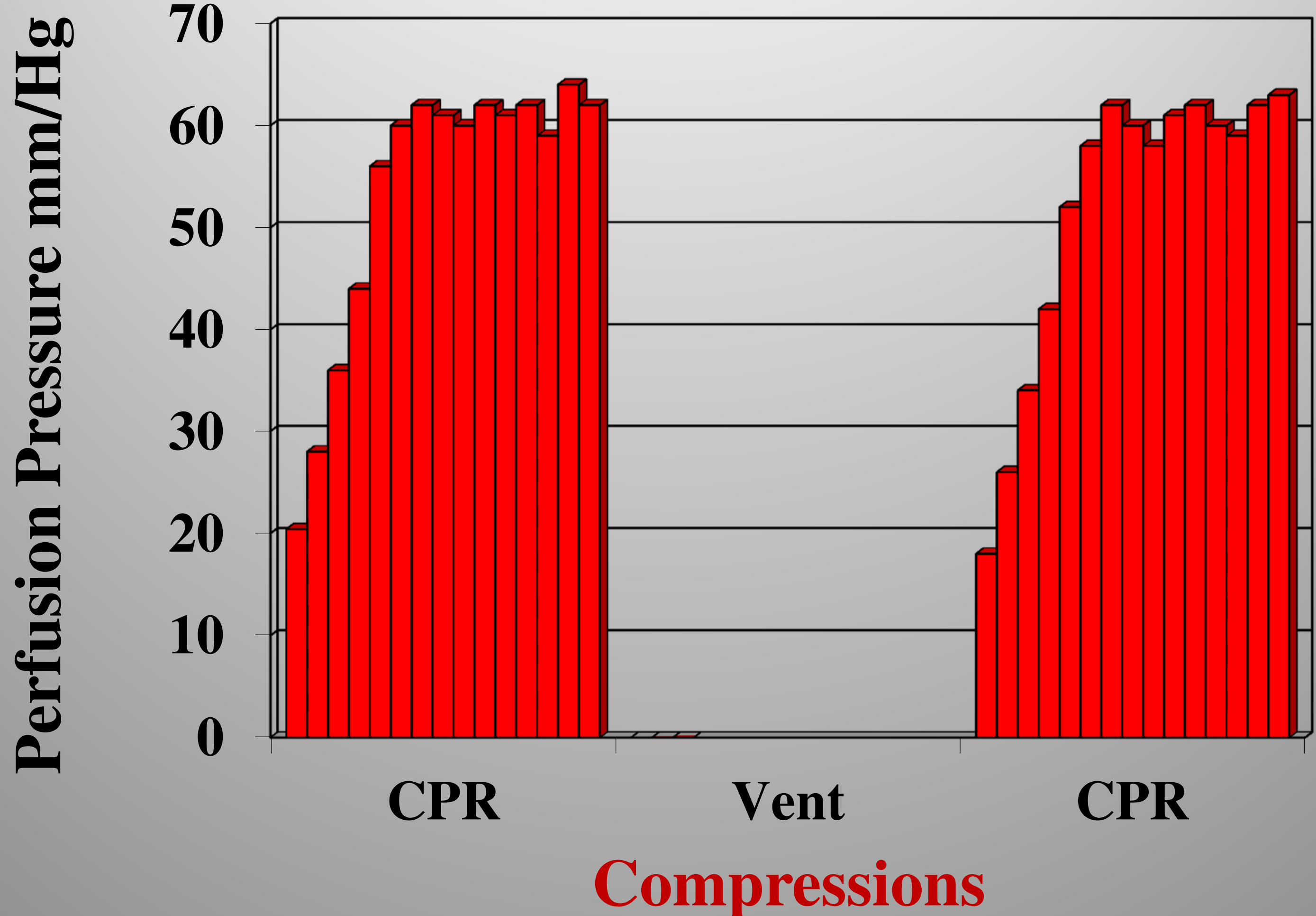
Positive Pressure Ventilation



Passive Exhalation



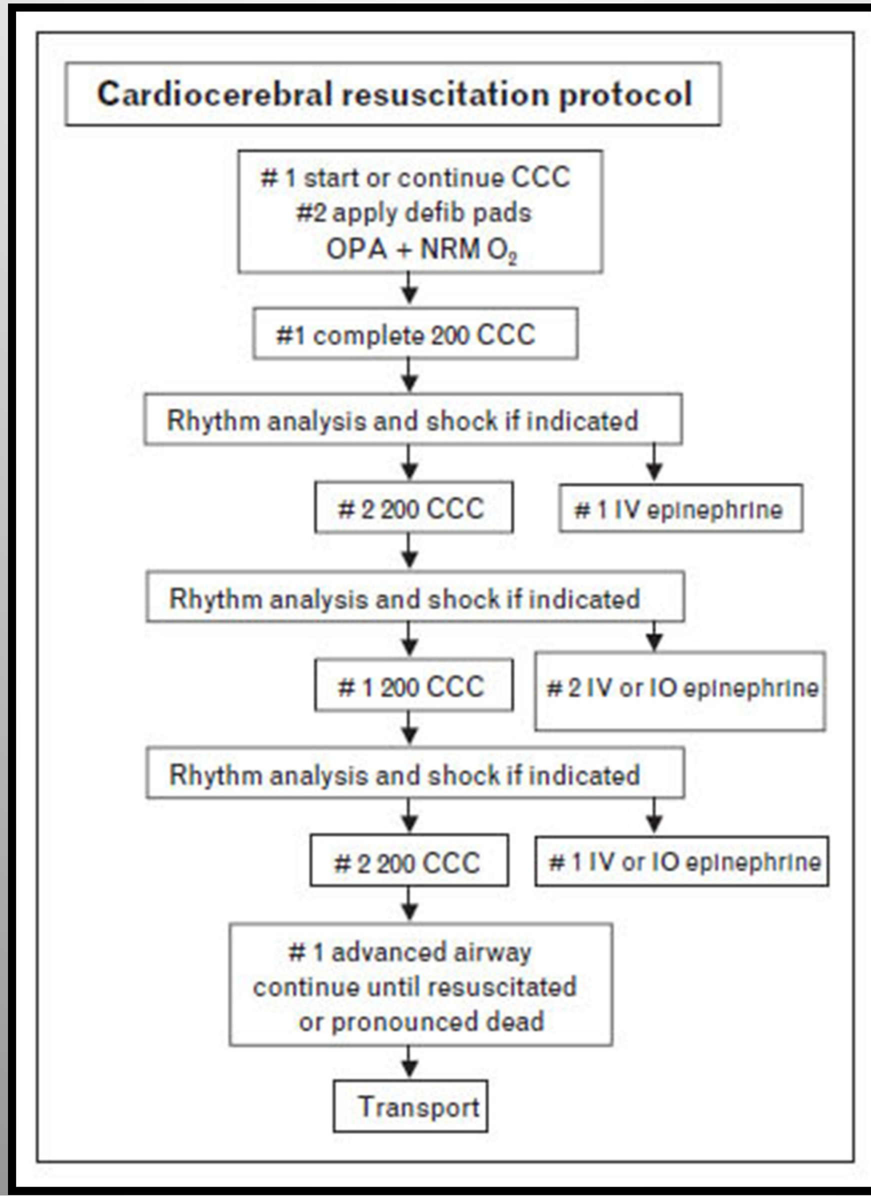
Chest Compressions: Perfusion Pressure



Cardiocerebral Resuscitation (CCR) AKA Continuous Chest Compression CPR (CCC CPR)

- **For Bystander Witnessed Cardiac Arrest**
- **Presumed to be Primary Cardiac Arrest**
- **Ventilation not important in early management**
- **Blood is oxygenated but not being circulated**
- **Adequate oxygenation can be maintained with passive O_2 insufflations via open airway and 100% O_2 via mask**
- **Emphasis on continuous compressions and no positive pressure ventilation for first 8 minutes**

Cardiocerebral Resuscitation



Clinical Trials of CCR

Kellum MJ, Kennedy KW, Barney R, et al Cardiocerbral Resuscitation Improves Neurologically Intact Survival of Patients With Out-of-Hospital Cardiac Arrest. Ann Emerg Med 2008;52: 244-52

Design: Before-and after observational study of the effect of a new protocol on survival from OHCA 2 rural County EMS Systems in Wisconsin

Results:	2001-2003		2004-2006	
# subjects	92		89	
Survivors	18	20%	42	47%
CPC=1	14	15%	35	39%

Clinical Trials of CCR

Bobrow BJ, Clark LL, Ewy GA, et al. Minimally Interrupted Cardiac Resuscitation by Emergency Medical Services for Out-of-Hospital Cardiac Arrest. JAMA.2008; *299(10):1158-1165*

Design: Prospective observational study of the effect of a new minimally interrupted CPR protocol on survival from OHCA. 2 Metropolitan EMS Systems Arizona beween Jan 1 2005 and Nov 30, 2007. Before and after initiation of Minimally interrupted chest compression training

Results:	Before		After		OR(95% CI)
# subjects	218		668		
Survivors	4	1.8%	36	5.4%	3.0 (1.1-8.9)
Survive Witnessed Vfib	2/43	4.7%	23/131	17.6%	8.6 (1.8-42)

Clinical Trials of CCR

Mosier J, Itty A, Sanders A, et al. Cardiocerebral Resuscitation Is Associated With Improved Survival and Neurologic Outcome from Out-of-hospital Cardiac Arrest in Elders. ACADEMIC EMERGENCY MEDICINE 2010; 17:269–275

Design: Retrospective analysis of data Save Hearts in Arizona Registry Jan 2005-Sep 2008

Results:	CCR	Std ACLS	OR(95% CI)
# subjects	1,024	2,491	
Survivors	96 9.4%	108 4.3%	3.0 (2.05-4.49)
CPC+1-2	57/59 96.6%	75/88 85.2%	6.5 (1.31-32.8)

Key Points

• In spite of four decades of standards and guidelines, the survival of patients with out-of-hospital cardiac arrest (OHCA) remains dire. One major contributor to the ineffectiveness of the guidelines approach is that they advocated the same approach to two entirely different pathophysiologic conditions: cardiac arrest and respiratory arrest.

• In initial clinical trials, Cardio Cerebral Resuscitation (CCR) or Continuous Chest Compression CPR (CCC CPR) results in better outcomes than standard CPR in patients with primary cardiac arrest. CPR may be better (but not ideal) for patients with respiratory arrest.

Is Advanced Airway Management Beneficial in OHCA ?

Hasegawa K, Hiraide A, Chang Y, Brown DFM. Association of Prehospital Advanced Airway Management With Neurologic Outcome and Survival in Patients With Out-of-Hospital Cardiac Arrest. JAMA. 2013;309(3):257-266

Design: Prospective, nationwide, population-based study (All-Japan Utstein Registry) involving 649,654 consecutive adult patients who had an OHCA and in whom resuscitation was attempted by emergency responders with subsequent transport to medical institutions from Jan. 2005 through Dec. 2010.

Results:

	BVM	Advanced	
#Subjects	367,837(56.7%)	281,522(43.4%)	
		ETI	Supra Glotic
		41,972(6.5%)	239,550(36.9%)

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Results:	BVM	Advanced	OR(95%CI)
ROSC	25,904 (7%)	16,299(5.8%)	0.81(0.79-0.83)
Unadjusted			
Adjusted			0.67(0.66-0.69)

Results:	BVM	ETI	OR(95%CI)
ROSC	25,904 (7%)	3,514(8.4%)	1.21(1.16-1.25)
Unadjusted			
Adjusted			0.86(0.82-0.89)

Results:	BVM	Supra Glotic	OR(95%CI)
ROSC	25,904 (7%)	12,785(5.3%)	0.74(0.73-0.76)
Unadjusted			
Adjusted			0.64(0.62-0.65)

Is Advanced Airway Management Beneficial in OHCA ?

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Results:	BVM	Advanced	OR(95%CI)
CPC 1-2 Unadjusted	10,759 (2.9%)	3,156(1.1%)	0.38(0.36-0.39)
Adjusted			0.38(0.37-0.40)

Results:	BVM	ETI	OR(95%CI)
CPC 1-2 Unadjusted	10,759 (2.9%)	432(1.0%)	0.35(0.31-0.38)
Adjusted			0.41(0.37-0.45)

Results:	BVM	Supra Glotic	OR(95%CI)
CPC 1-2 Unadjusted	10,759 (2.9%)	2,724(1.1%)	0.38(0.37-0.40)
Adjusted			0.38(0.36-0.40)

Is Advanced Airway Management Beneficial in OHCA ?

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Adjusted for a predefined set of potential confounders including: age, sex, cause of cardiac arrest, first documented rhythm, bystander witnessed, type of cardiopulmonary resuscitation (CPR) initiated by bystander, use of a public access automated external defibrillator by bystander, epinephrine administration, time from receipt of call to CPR by emergency medical service, and time from receipt of call to hospital arrival.

Key Points

- The results of the CCR studies support the concept that aggressive airway management is not important in the initial management of OHCA. The findings of this study are consistent with several studies in trauma and pediatric patients. These studies have suggested that prehospital endotracheal intubation may lead to a decreased rate of favorable neurological outcome.

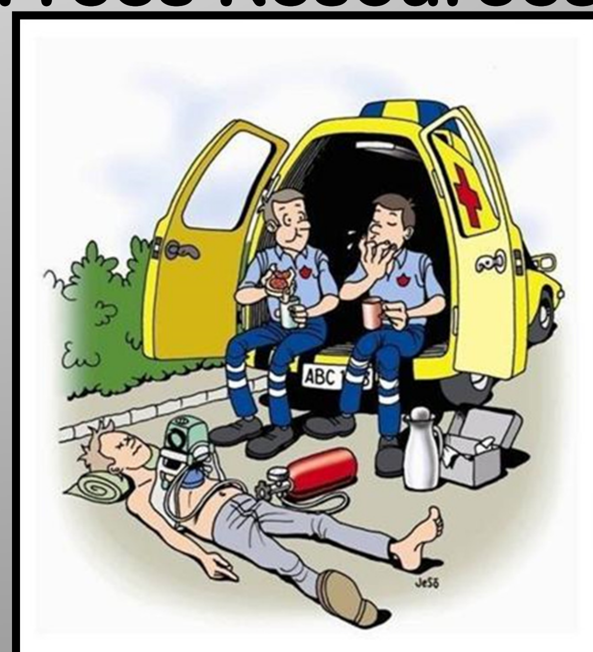
- This observational study contradicts the assumption that aggressive airway intervention is associated with improved outcomes and provide an opportunity to reconsider the approach to prehospital airway management in OHCA.

CPR Adjuncts

LUCAS Device

LUCAS Device

- Good CPR is Difficult
- Rescuer Fatigue → Poor Quality CPR
- Increased Resource Utilization
- LUCAS Device Consistently Delivers 2” Compression, full recoil, @100/min.
- Better Quality CPR and Frees Resources for other tasks
- Improves Outcome



LUCAS Device vs. Manual CPR in Out-of Hospital Cardiac Arrest

Mechanical Chest Compressions and Simultaneous Defibrillation vs Conventional Cardiopulmonary Resuscitation in Out-of-Hospital Cardiac Arrest: The LINC Randomized Trial. JAMA Published Online November 17, 2013

Design: Randomized Multicentre Trial in 4 Swedish, 1 British, and 1 Dutch EMS Systems (Jan 2008- Aug 2012)

Results:	LUCAS		Manual		p=
# Enrolled	1300		1289		
4hr. Survival	307	23.6%	305	23.7%	>0.99
ROSC	460	35.4%	446	34.6%	0.68
6 Month CPC 1-2	110	8.5%	98	7.6%	0.43

Key Points

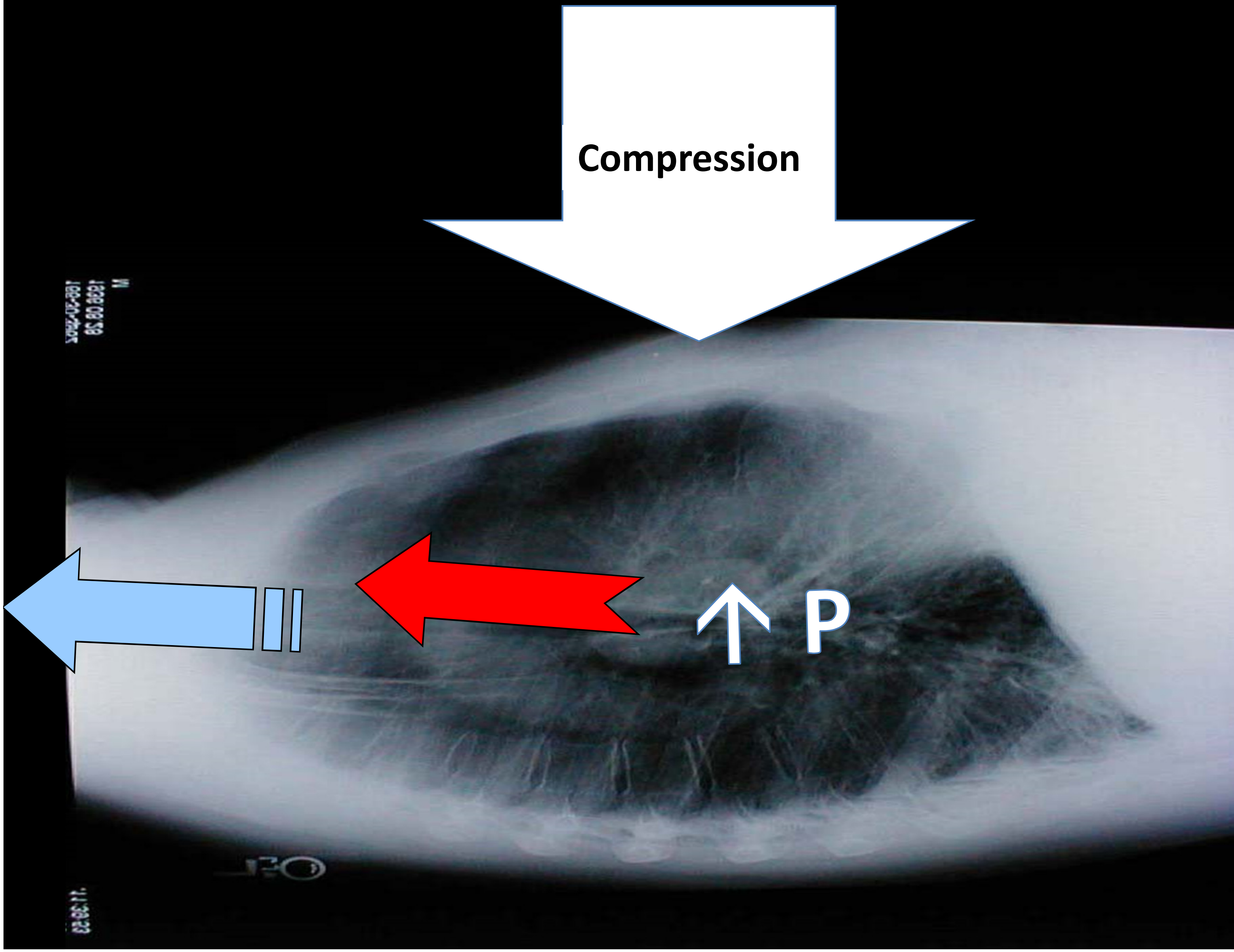
• Lucas Device or other automated chest compressors may decrease manpower requirements for resuscitation from OHCA

• Lucas Device may provide more consistent chest compressions than Manual CPR (rate, depth, and full recoil)

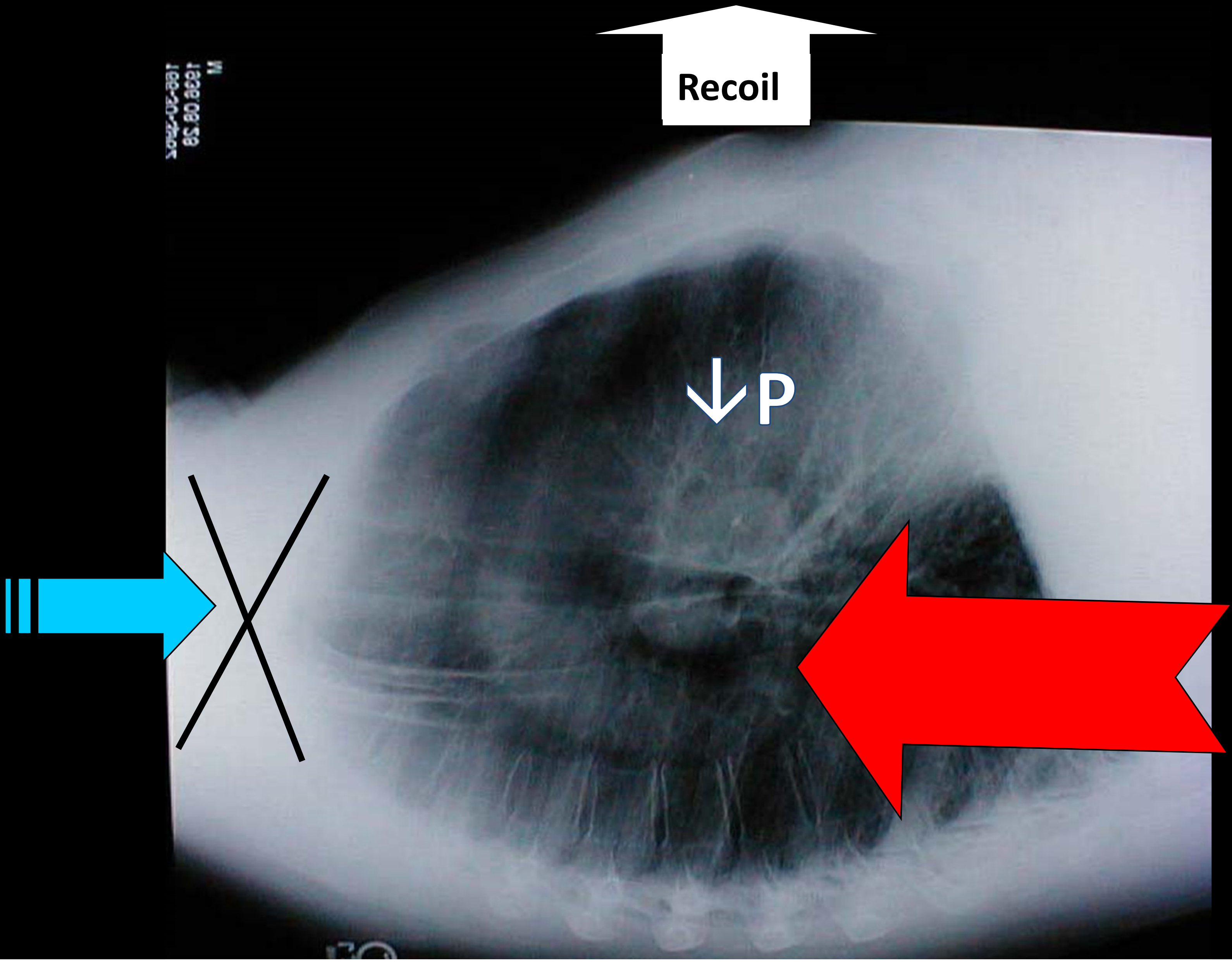
• To date no studies have demonstrated an improvement in clinically important outcomes associated with these devices. The only randomized study of Lucas Device vs Manual CPR in OHCA demonstrated no significant difference in survival or neurologic status of survivors.

Impedance Threshold Device

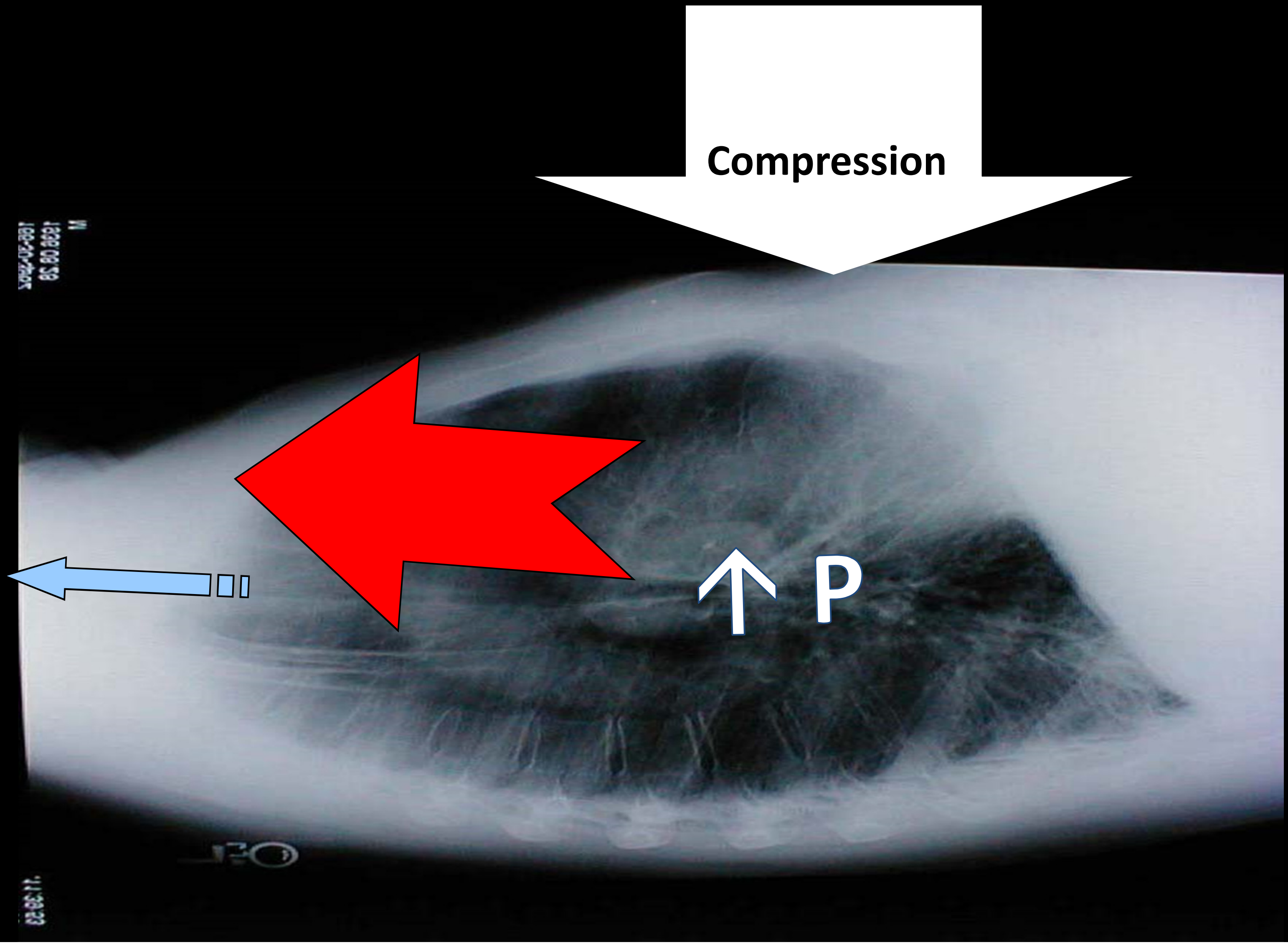
Impedance Threshold Device



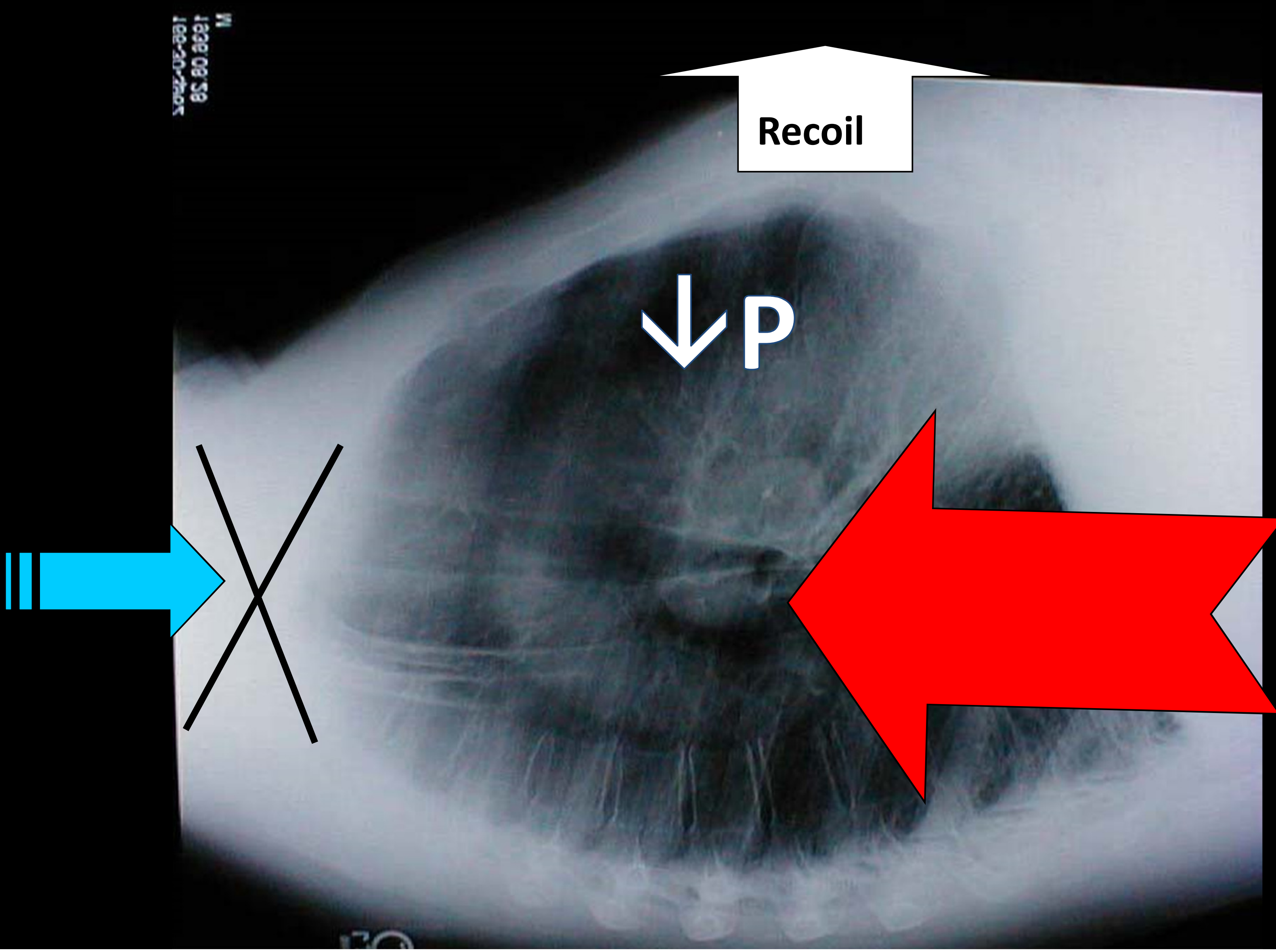
Impedance Threshold Device



Impedance Threshold Device



Impedance Threshold Device



A Trial of an Impedance Threshold Device in Out-of-Hospital Cardiac Arrest

Aufderheide TP, Nichol G, Rea TD, et al. A Trial of an impedance threshold device in out-of-hospital cardiac arrest. NEJM; 2011 365: 798-806

Design: ITD vs Sham Device Prospective Randomized Trial

Results:	Sham ITD		Active ITD		p=
#Enrolled	4,345		4,373		
ROSC:	1,206	27.8%	1,186	27.1%	0.51
Survive to D/C	355	8.2%	357	8.2%	0.99
Modified Rankin Score ≤ 3	260	6.0%	254	5.8%	0.71

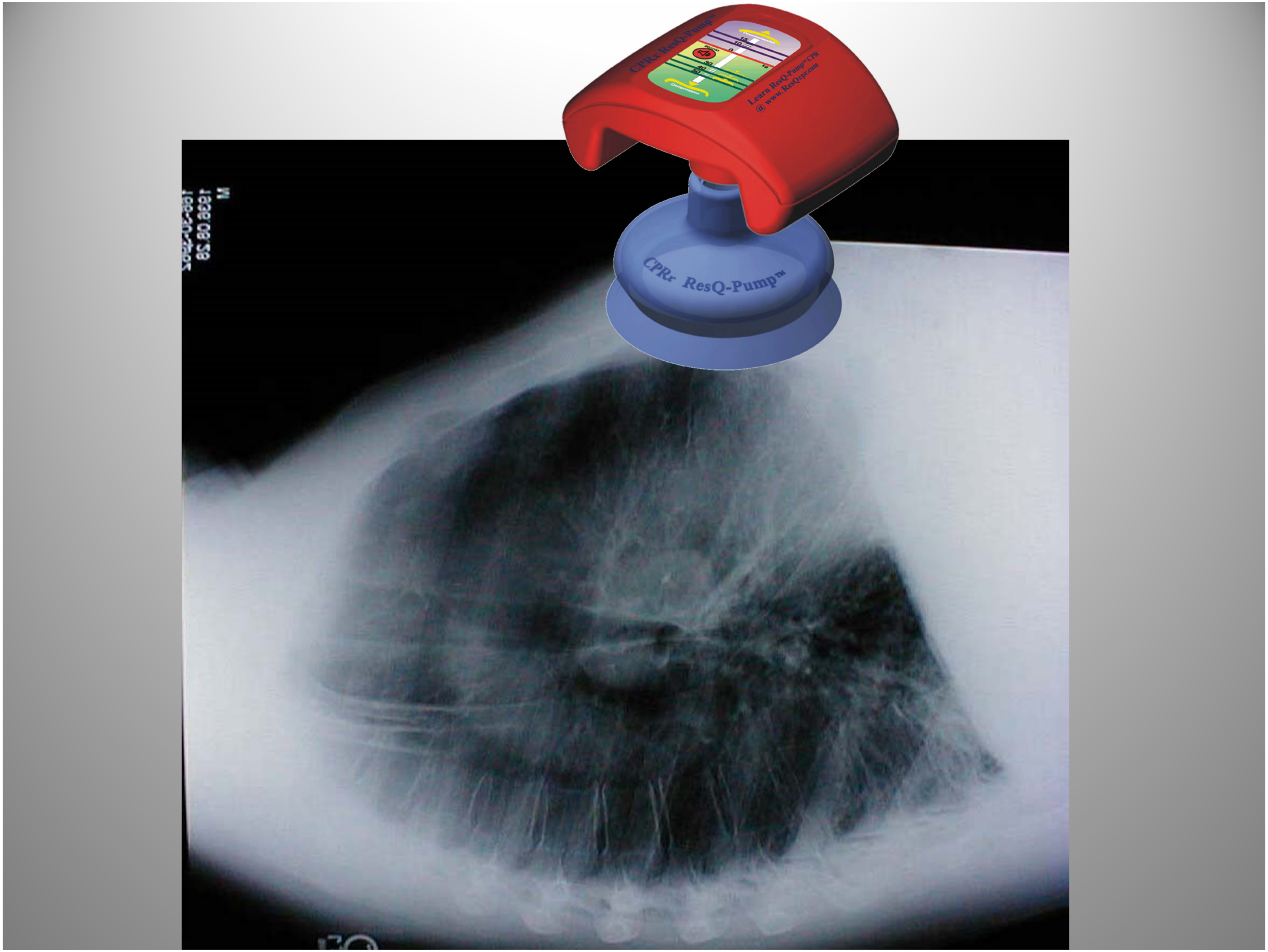
Key Points

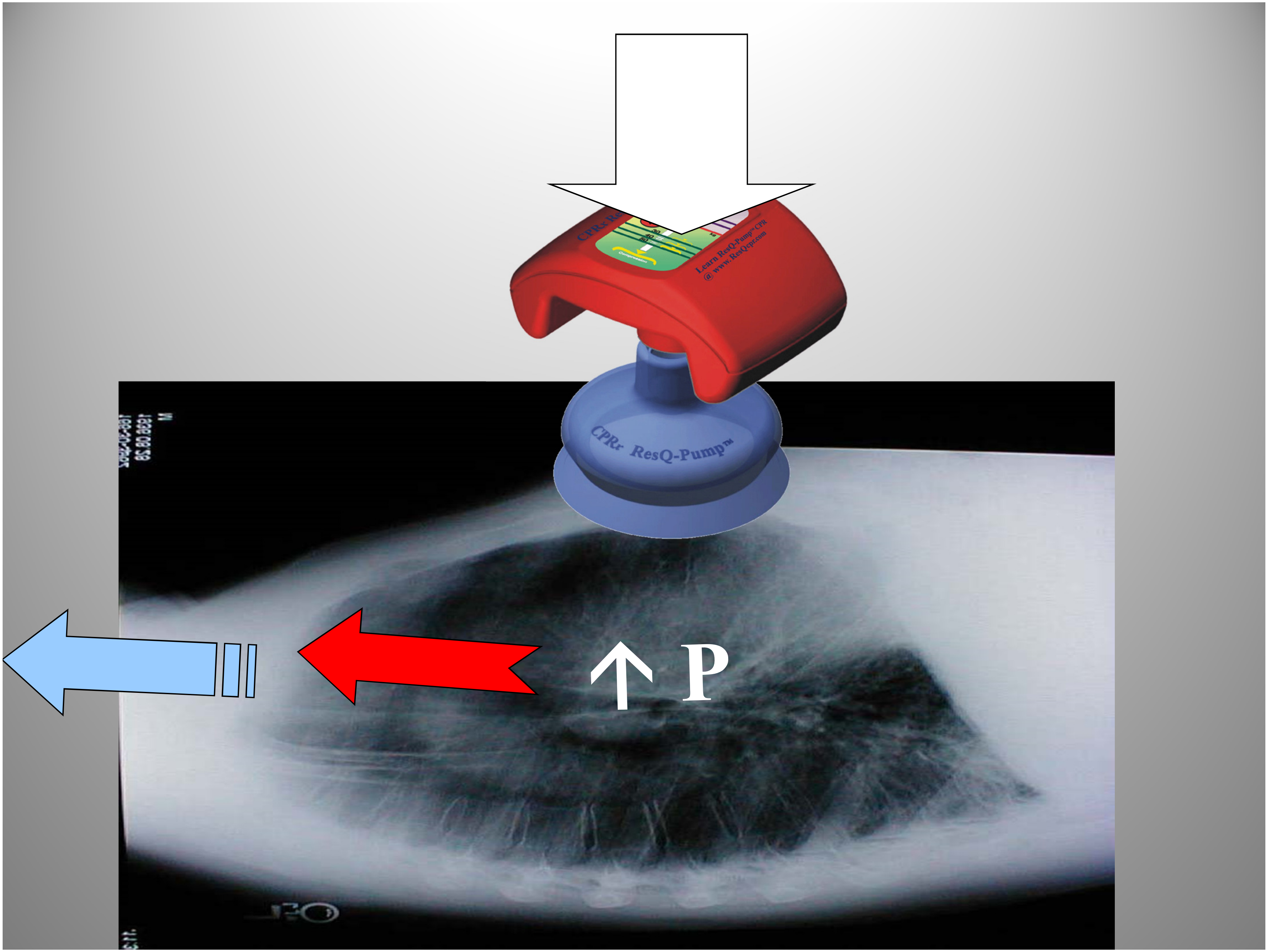
- Inspiratory Impedance threshold device (ITD) has been shown in animal studies to improve hemodynamics, vital organ perfusion and neurologically intact survival

- Small clinical trials have shown that ITD can increase aortic pressure during the chest compression phase of CPR and increase short term survival in victims of OHCA

- To date no studies have demonstrated an improvement in clinically important outcomes associated with these devices. The only randomized study of ITD in OHCA demonstrated no significant difference in survival or neurologic status of survivors.

Active Compression Decompression Device


CPRx ResQ-Pump™
Decompression
15
10
0
90/min
30
40
50
kg
Compression
Learn ResQ-Pump™ CPR
@ www.ResQcpr.com
CPRx ResQ-Pump™


CPRx ResQ-Pump™
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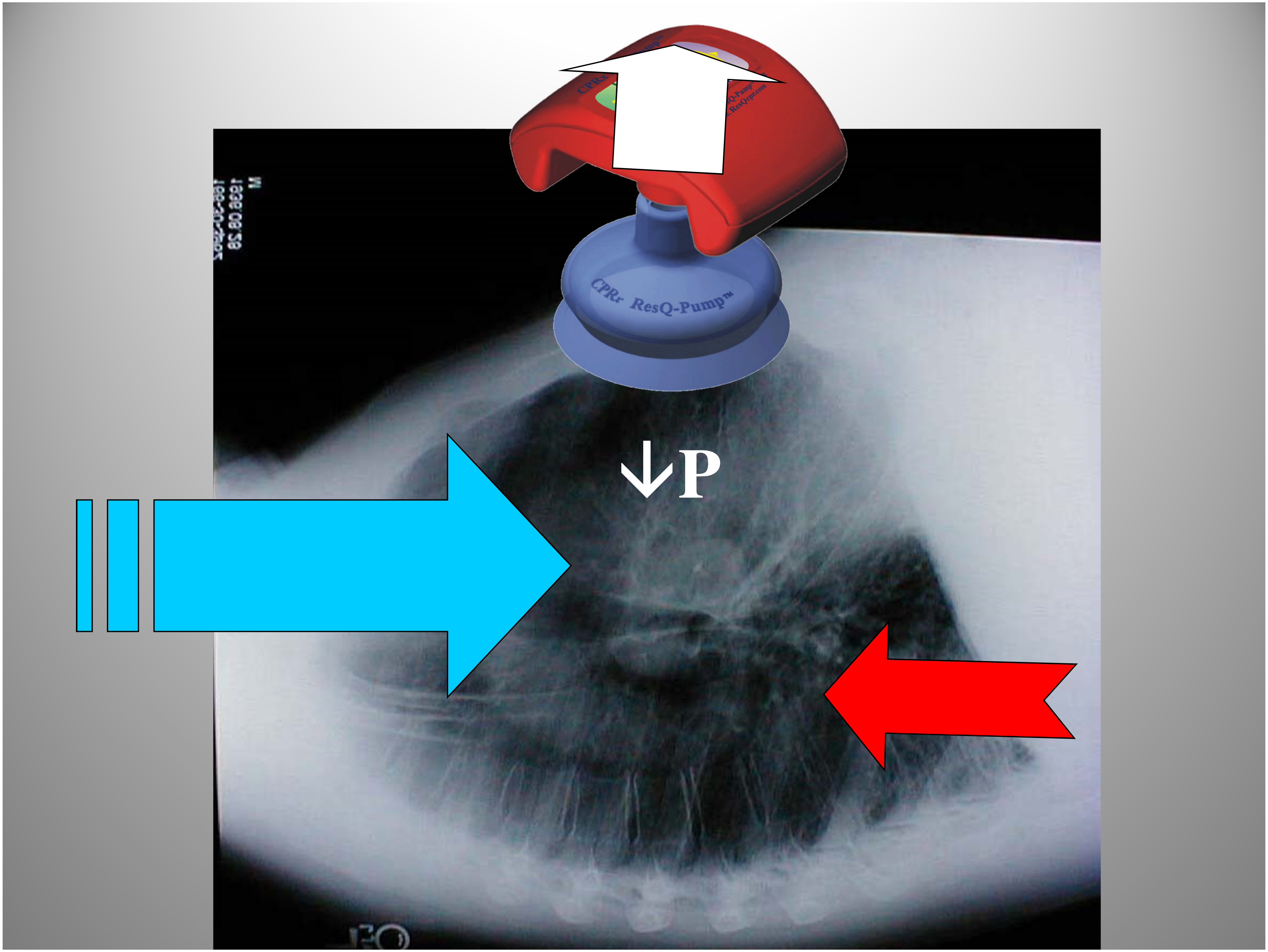
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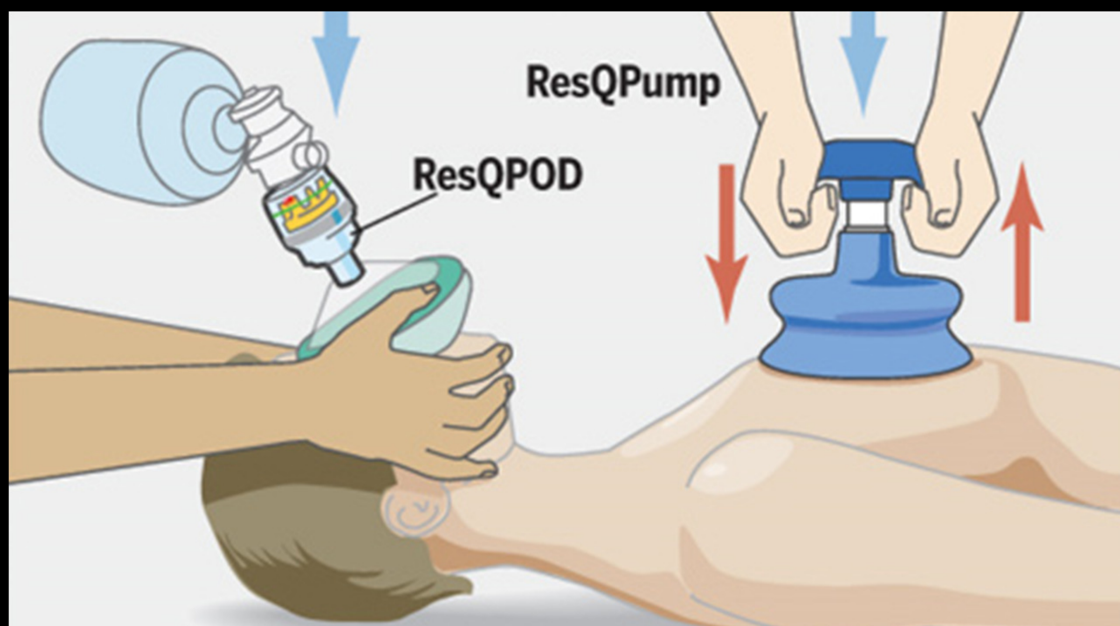
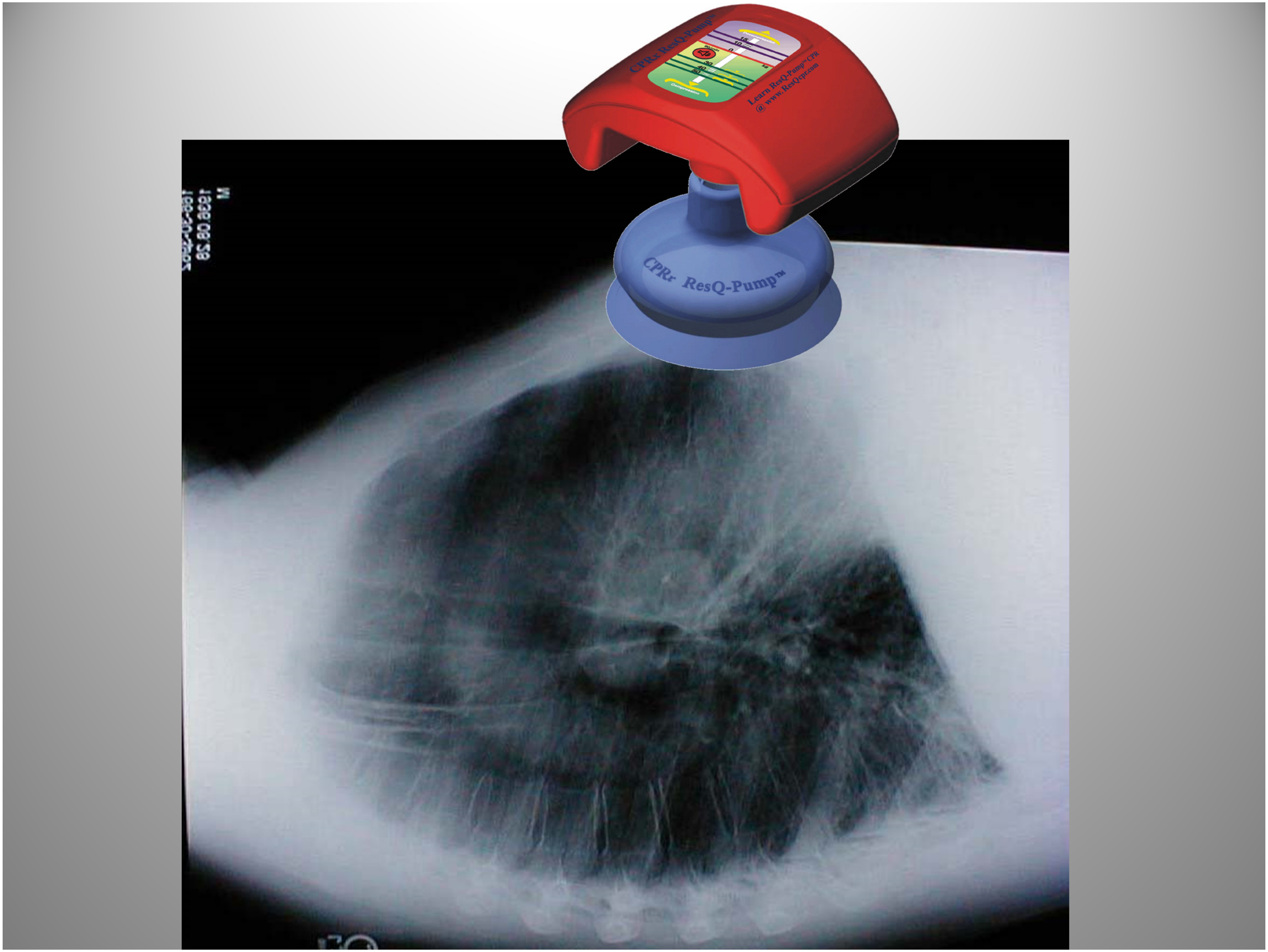
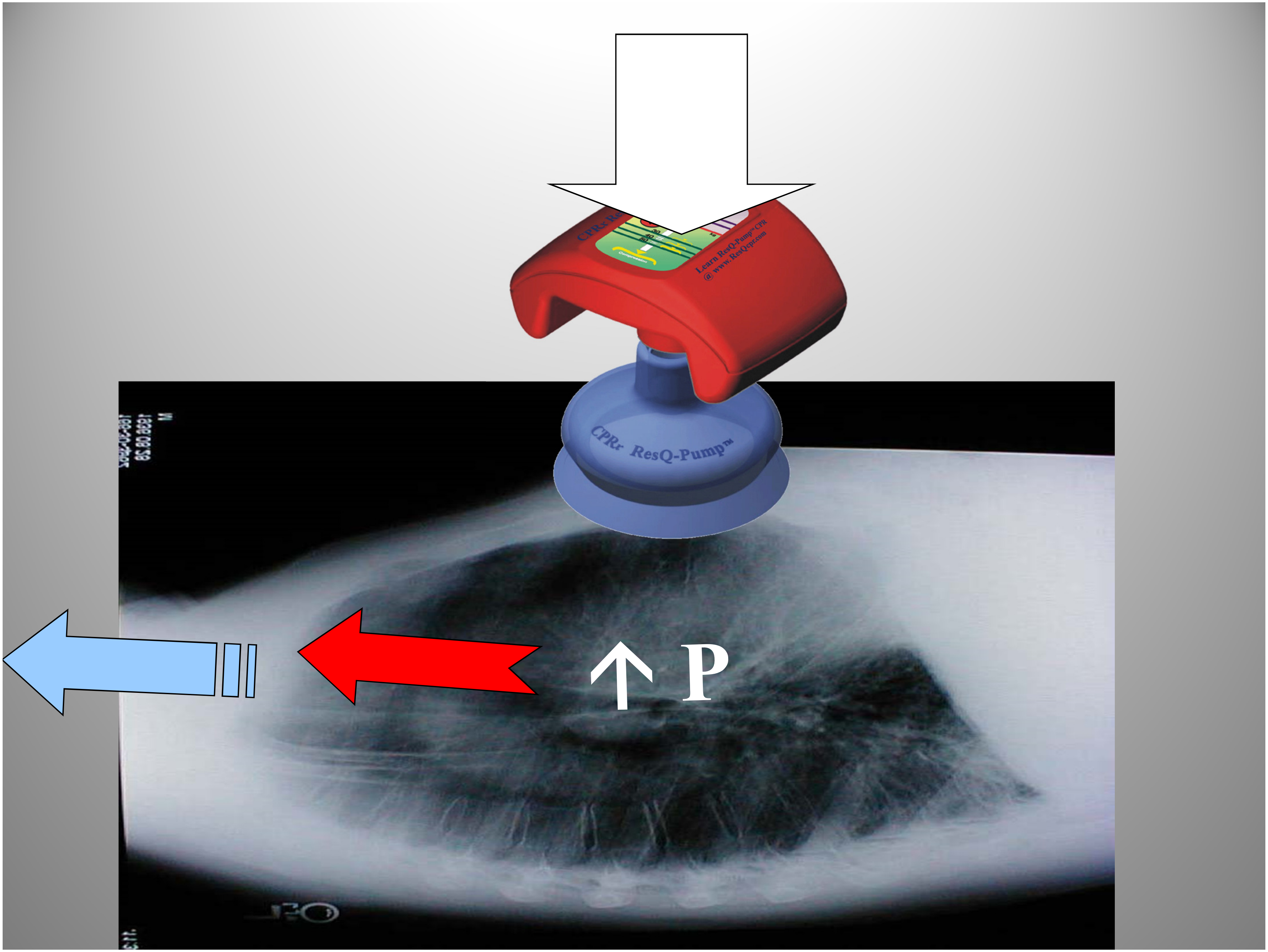
Active Compression Decompression Device
+
Inspiratory Impedance Threshold Device




CPRx ResQ-Pump™
Learn ResQ-Pump™ CPR
@ www.ResQcpr.com
CPRx ResQ-Pump™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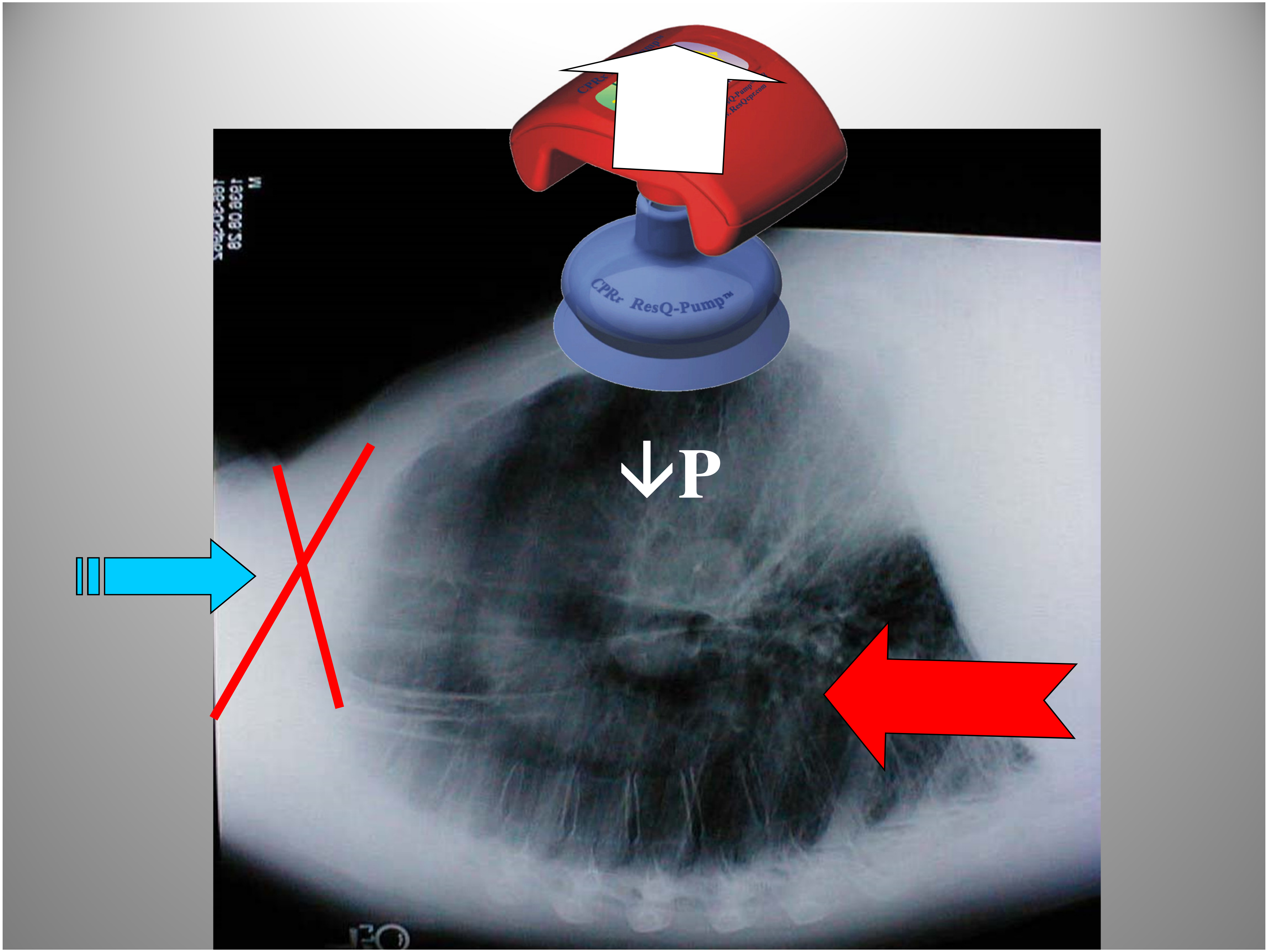

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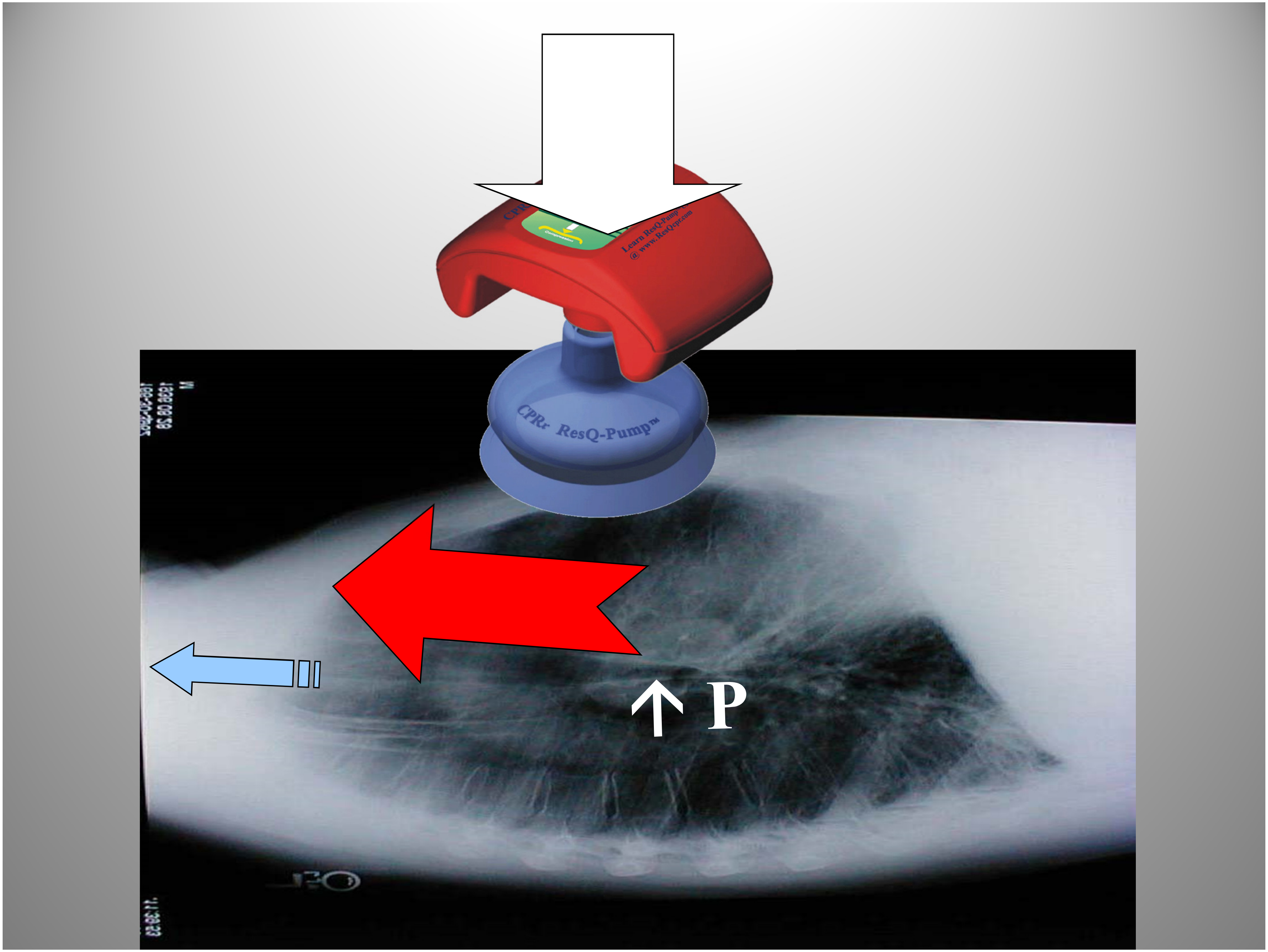
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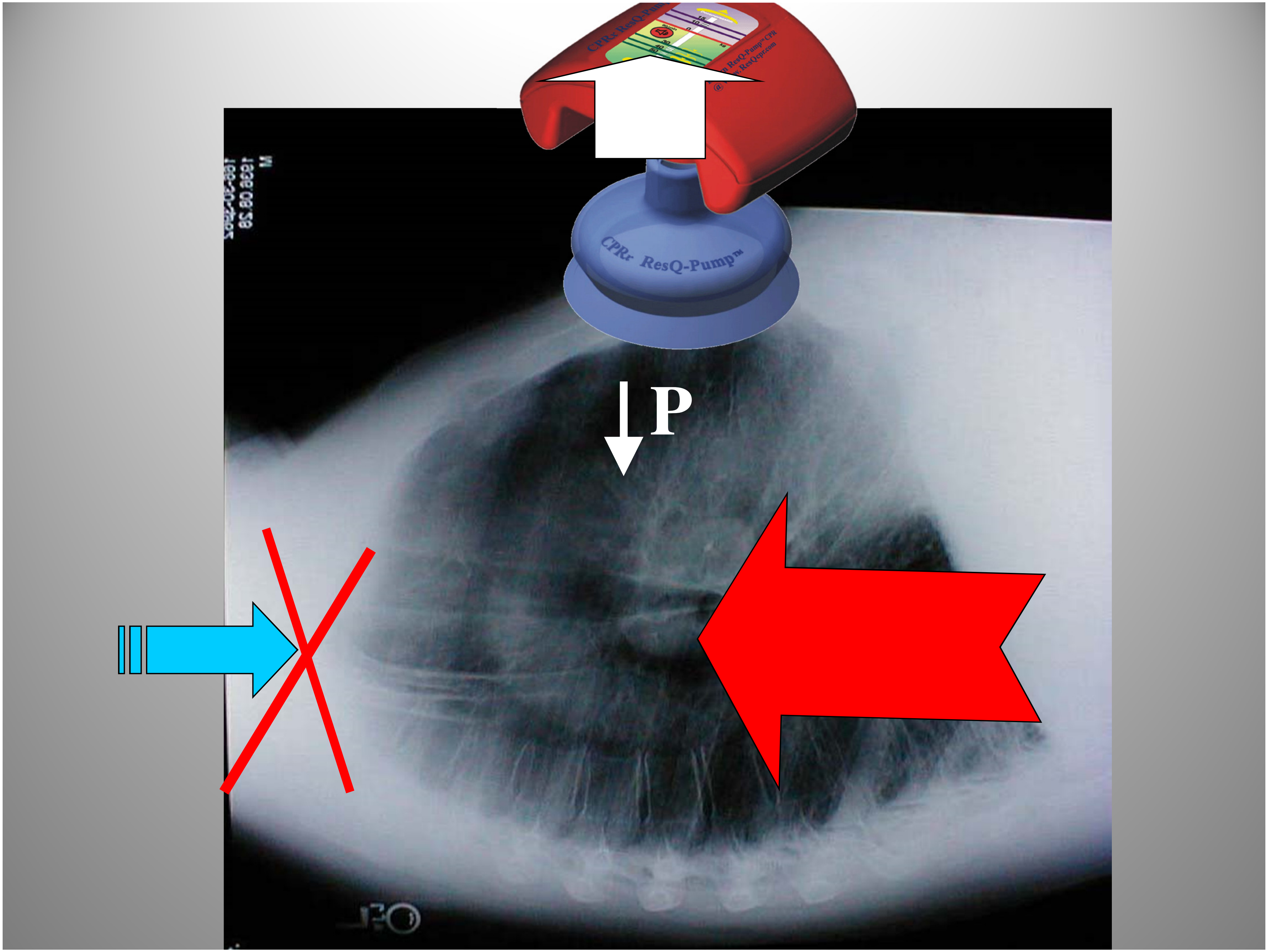
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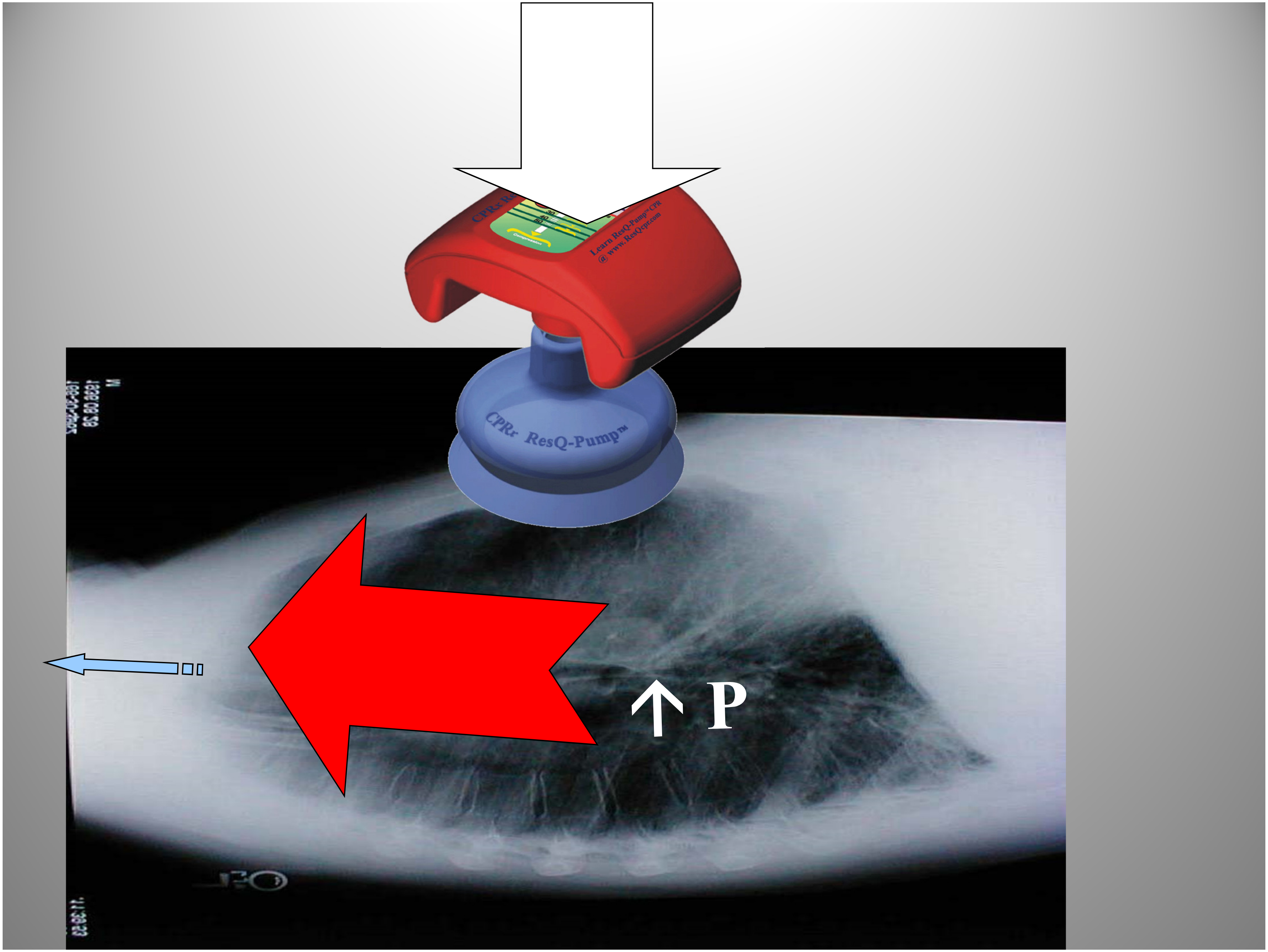
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Learn ResQ-Pump™ CPR
@ www.ResQcpr.com
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Clinical Trial of ACDC CPR plus ITD to Standard CPR

Aufderheide TP, Frascone RJ, Wayne MA, et al. Standard cardiopulmonary resuscitation versus active compression-decompression cardiopulmonary resuscitation with augmentation of negative intrathoracic pressure for out-of-hospital cardiac arrest: a randomised trial. ***Lancet 2011; 377: 271-352***

Design: Randomized Controlled trial comparing Std CPR to ACDC CPR with ITP. Primary end point= survival with CPC ≤3

Results:	Std CPR	ACDC CPR	p=	OR (95% CI)
1° Cardiac				
#Enrolled	1201	1269		
# Subjects	813	840		
Survive CPC ≤3	47(5.8%)	75 (8.9%)	0.019	1.58(1.08-2.30)
Non Trauma				
# Subjects	1318	1396		
Survive CPC ≤3	75 (5.69%)	110 (7.88%)	0.027	1.42(1.04-1.95)

Key Points

- ACDC CPR in conjunction with an Inspiratory Impedance threshold device (ITD) has been shown in a large randomized clinical trial to improve survival and neurologic function from OHCA

- This randomized clinical trial demonstrated that ACDC CPR +ITD can increase survival with good neurologic function from OHCA due to a presumed cardiac etiology by 58% and from OHCA due to a variety of non-traumatic causes by 38.5% as compared to standard CPR

Therapeutic Hypothermia

Part 9: Post–Cardiac Arrest Care

2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Peberdy MA, Callaway CW, Neumar RW, Geocadin RG, et al. Part 9: post– cardiac arrest care: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation. 2010;122(suppl 3):S768 –S786.*

Comatose adult patients with ROSC after out-of-hospital VF cardiac arrest should be cooled to 32°C to 34°C (89.6°F to 93.2°F) for 12 to 24 hours **(Class I, LOE B).**

Induced hypothermia also may be considered for comatose adult patients with ROSC after in-hospital cardiac arrest of any initial rhythm or after out-of-hospital cardiac arrest with an initial rhythm of pulseless electrical activity or asystole
(Class IIb, LOE B).

Undetermined: How Soon, How Cold, How Long.

How Soon? How Cold? How Long?

How Soon?

- Brain hypothermia's mechanism of neuro-protection
  - Decrease metabolic demand
  - Prevent reperfusion injury
- Conjecture that the sooner the better

How Cold?

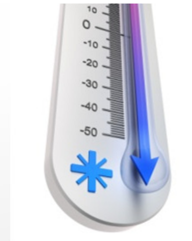
- Based on target temperatures of published trials
- Consensus 32^{o}-34^{o} C

How Long?

- Based on duration in published trials
- 12-24 hrs

ILCOR Recommendations
spawned a cooling-technology industry that aggressively markets Its products

How Cold???



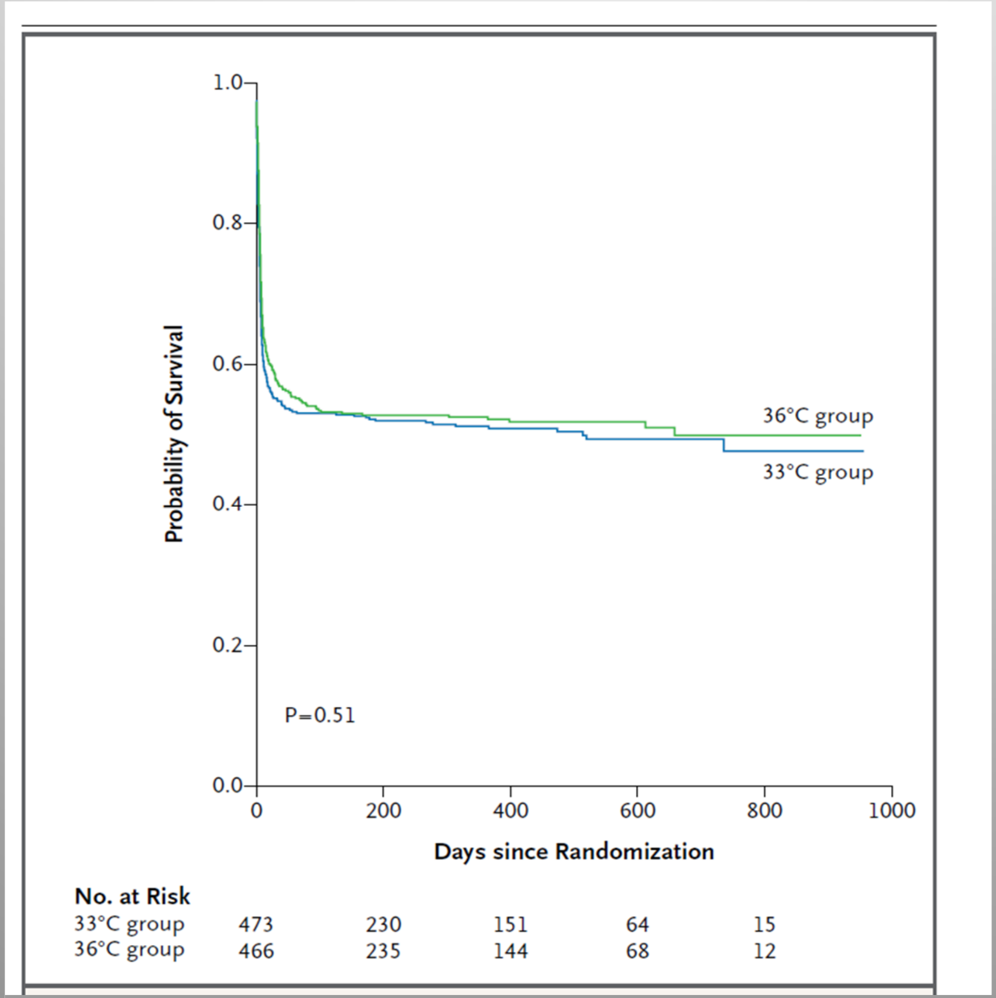
Nielsen N, Wetterslev J, Cronberg T, et al. Targeted Temperature Management at 33°C versus 36°C after Cardiac Arrest . NEJM. 2013; published online November 17, 2013

Design: International, multicenter study of unconscious OHCA of presumed cardiac etiology randomized to targeted temperature management at 33^{o} C vs. 36^{o} C

Results:	33^{o}	36^{o}	p=	OR(95% CI)
# Subjects	473	466		
1^{o} Outcome				
Death	235 (50%)	225 (48%)	0.51	1.06(0.89-1.28)
2^{o} Outcome				
CPC=3-5	251 (54%)	242 (52%)	0.78	1.02(0.88-1.16)
Modified Rankin Score 4-6	245 (52%)	239 (52%)	0.87	1.01(0.89-1.14)

How Cold???

Nielsen N, Wetterslev J, Cronberg T, et al. Targeted Temperature Management at 33°C versus 36°C after Cardiac Arrest . NEJM. 2013; published online November 17, 2013



Key Points

- This randomized prospective clinical trial demonstrated no benefit of lowering the body temperature to 33°C compared to actively maintaining it at 36°C
- There may be a clinically relevant benefit of controlling the body temperature at 36°C, instead of allowing fever to develop in patients who have been resuscitated after cardiac arrest.
- There is no evidence that targeting a body temperature of 33°C confers any benefit for unconscious patients admitted to the hospital after out-of-hospital cardiac arrest, as compared with targeting a body temperature of 36°C.

How Soon?

Due to the belief that the sooner therapeutic hypothermia is initiated following OHCA the better the outcome. Many EMS agencies have adopted strategies to begin cooling as soon as ROSC is achieved.

Others have initiated protocols to begin the cooling process during the resuscitation, so that cooling has already begun at the time of ROSC

Prehospital Induced Therapeutic Hypothermia

Kim F, Nichol G, Maynard C, et al. Effect of Prehospital Induction of Mild Hypothermia on Survival and Neurological Status Among Adults With Cardiac Arrest: A Randomized Clinical Trial. JAMA. 2013; Published online November 17, 2013

Design: Randomized clinical trial that assigned adults with prehospital cardiac arrest to standard care with or without prehospital cooling, accomplished by infusing up to 2 L of 4℃ normal saline as soon as possible following ROSC

Results:	V-Fib 583			Other 776		
	Cooled	Not Cooled	p=	Cooled	Not Cooled	p=
# Subjects	292	291		396	380	
Alive	183(62.7%)	187(64.3%)	0.69	76 (19.2%)	62(16.3%)	0.30
CPC=1-2	168(57.5%)	180(61.9)	0.69	57 (14.4%)	51(13.4%)	0.30

Prehospital Induced Therapeutic Hypothermia

Kim F, Nichol G, Maynard C, et al. Effect of Prehospital Induction of Mild Hypothermia on Survival and Neurological Status Among Adults With Cardiac Arrest: A Randomized Clinical Trial. JAMA. 2013; Published online November 17, 2013

Safety Data	Cooled	Not Cooled	p=
Rearrest post randomization	176 (26%)	138 (21%)	0.008
Pulmonary edema 1st CXR	256 (41%)	184 (30%)	<0.001
Diuretics first 12-48 hours	151 (23%)	109 (17%)	0.01
Glucose > 300 mg/dl	168 (25%)	208 (32%)	0.004

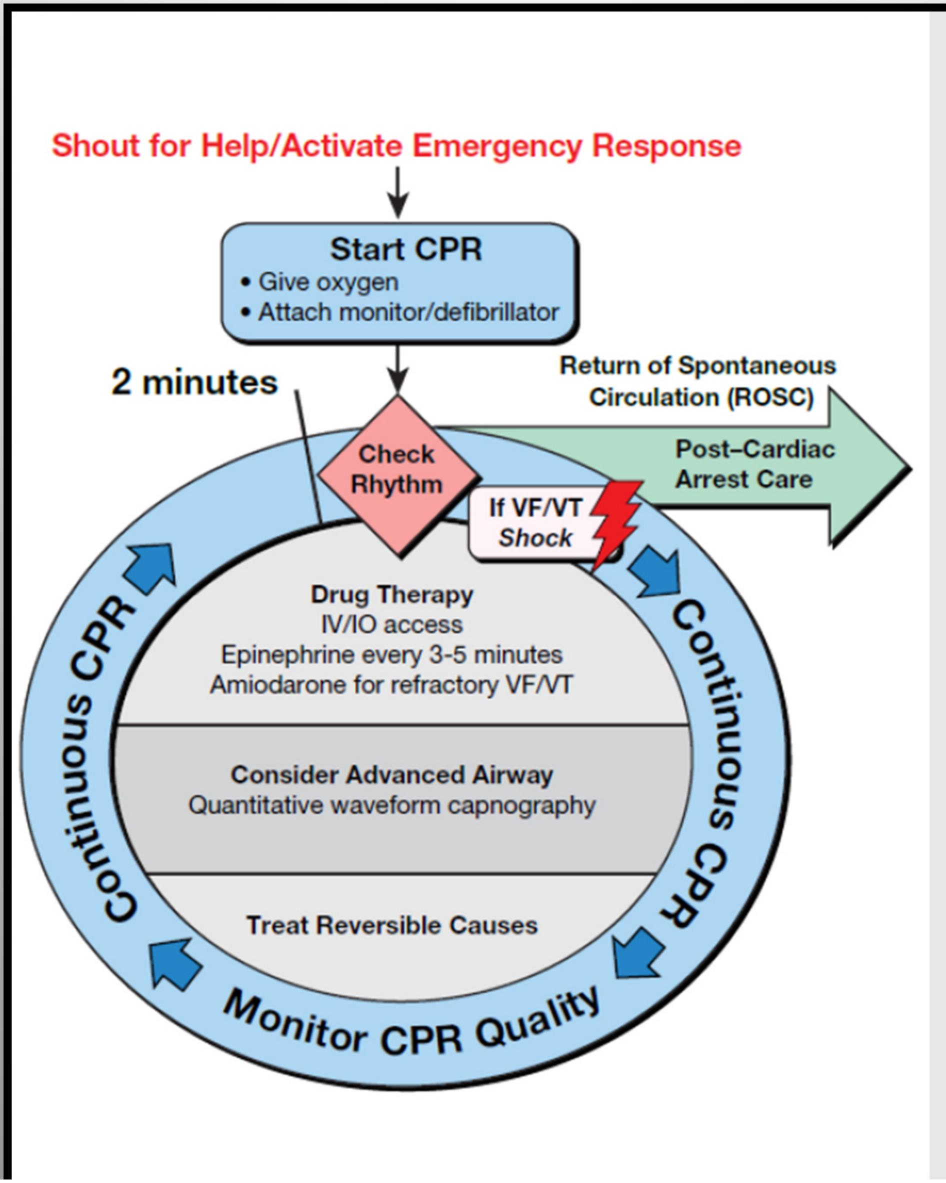
Key Points

- Early out-of-hospital cooling by rapid infusion with 2L 4°C normal saline reduced core temperature by more than 1°C and reduced the time to achieve the therapeutic temperature goal of 34°C by more than 1 hour.

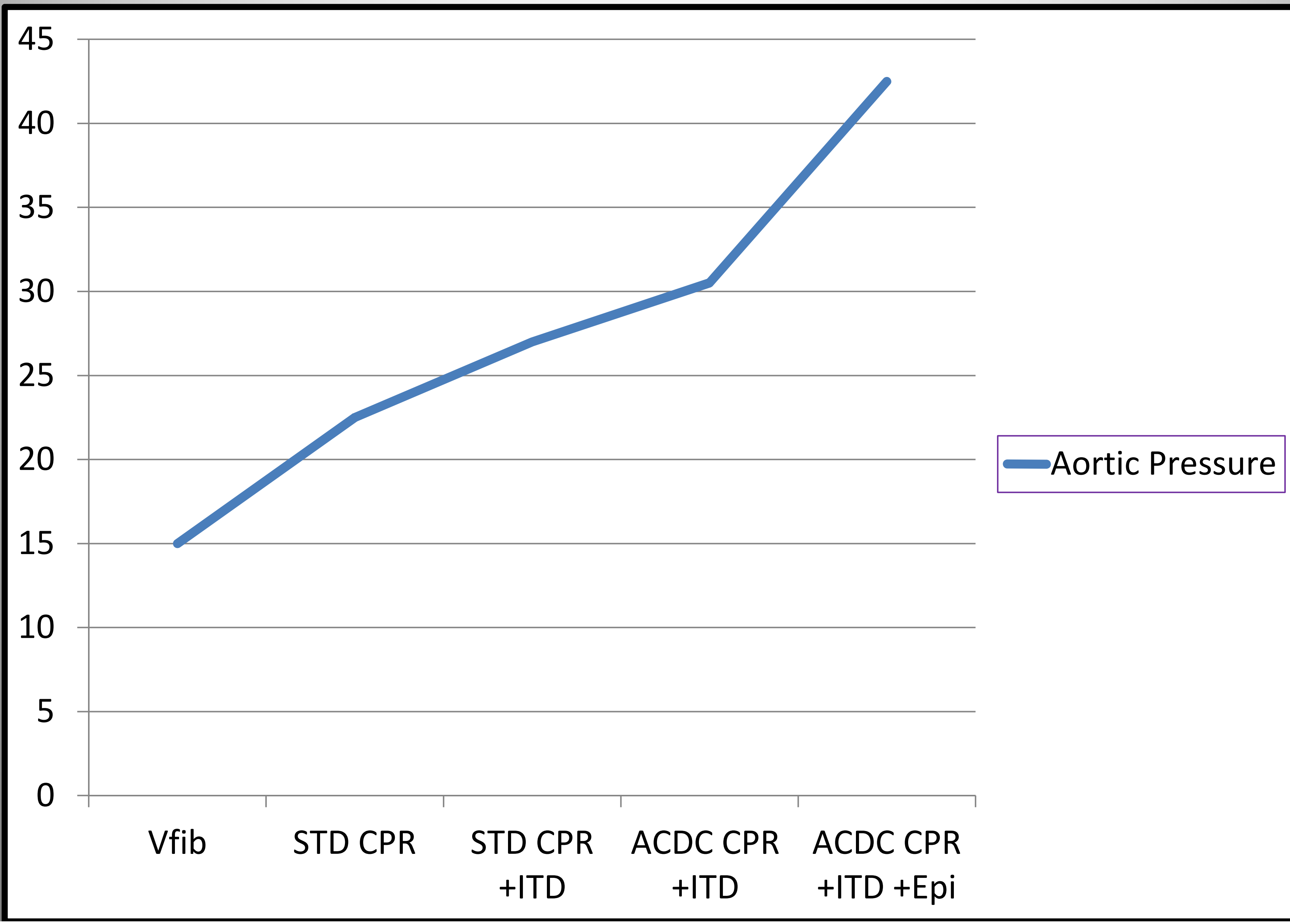
- Rapid fluid administration was associated with higher rates of rearrest during transport and increased transient pulmonary edema, which resolved within the first 24 hours.

- Although induced hypothermia may be a promising strategy to improve resuscitation and brain recovery following cardiac arrest, the results of this randomized clinical trial do not support the routine use of cold intravenous fluid in the prehospital setting to improve clinical outcomes.

Drugs = Epinephrine + Amiodarone



Epinephrine



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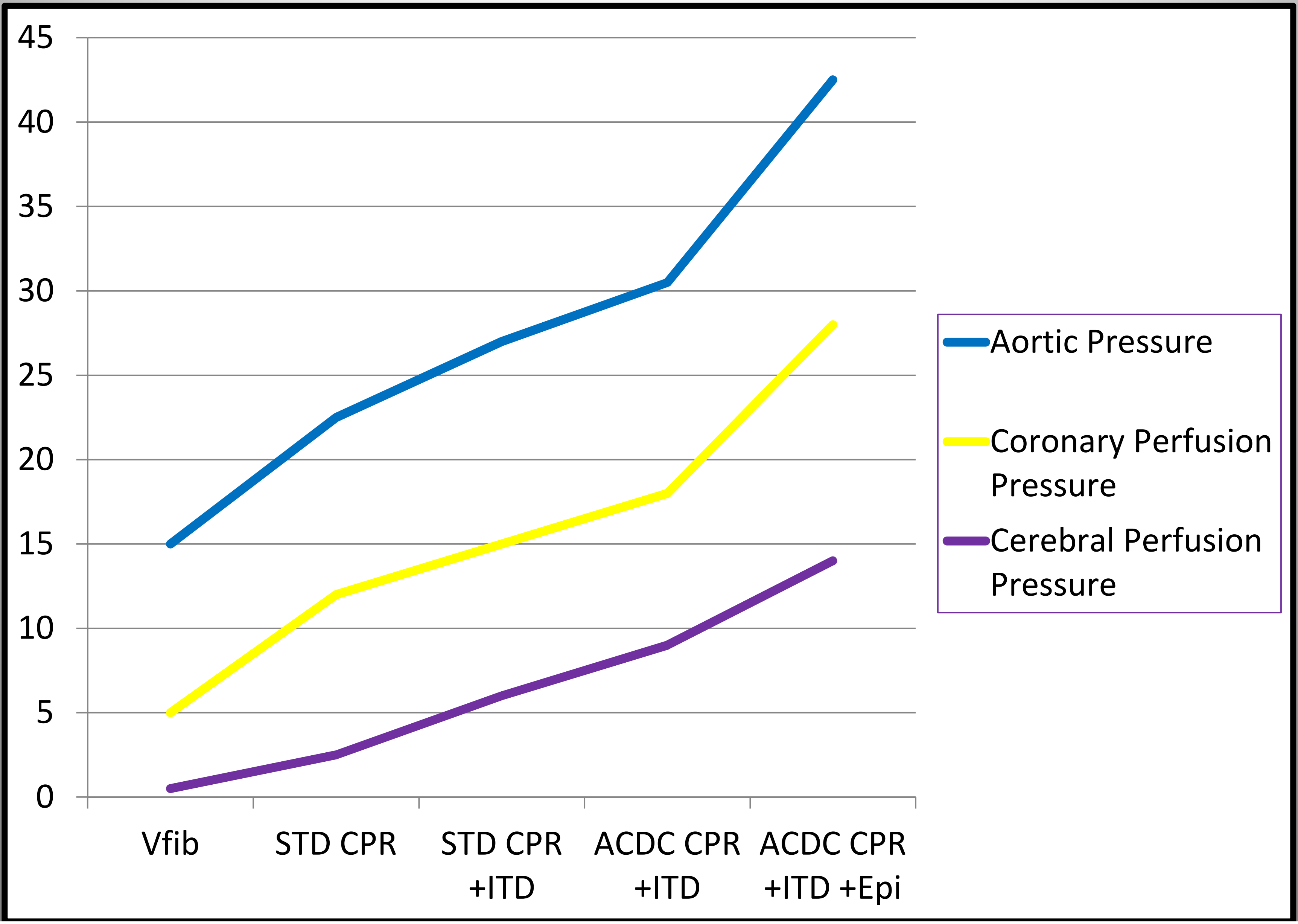

Year/Author	Design	Number of Subjects	ROSC OR	Survive OR	CPC 1, 2 OR
2012 Hagihara Japan	Observation Propensity Matched	13,401	2.36 (CI 2.22-2.50) P< 0.001	0.54 (CI 0.43-0.68) P< 0.001	0.21 (CI 0.10 -0.44) P< 0.001

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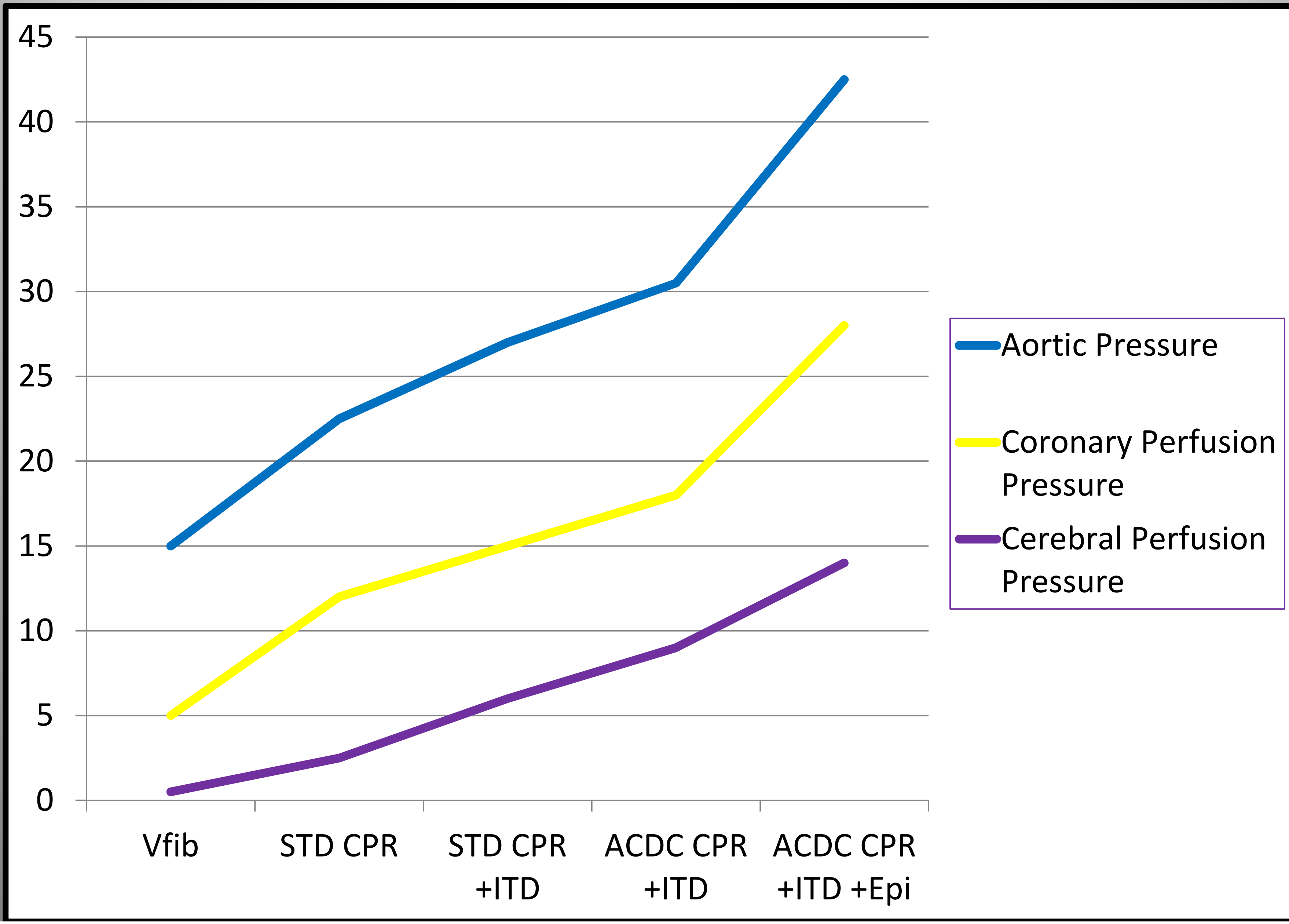
2002 Holmberg	Prospective Observational Cohort	10,966		Epi 3.4% No Epi 6.3%	

Epinephrine



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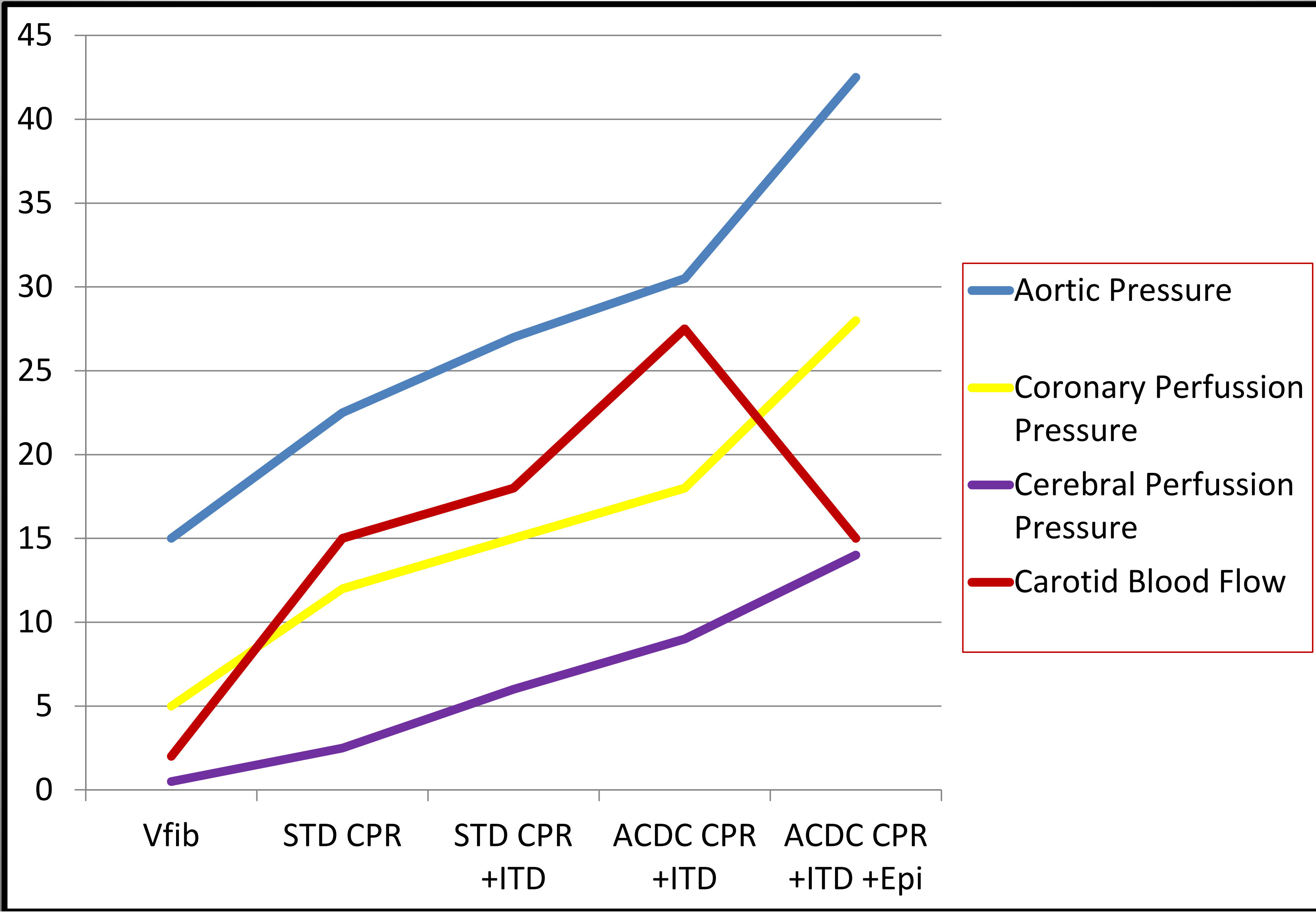
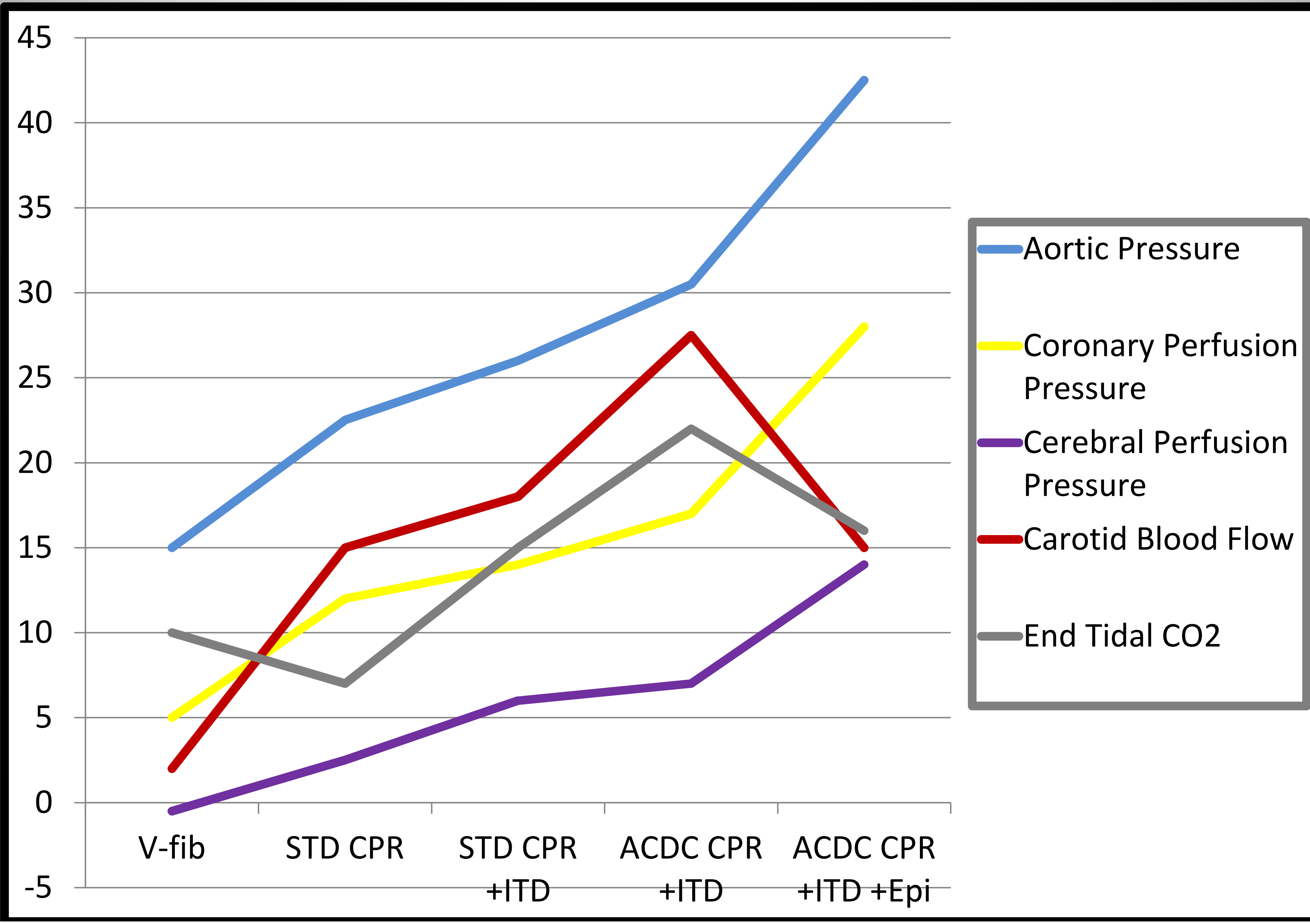


Epinephrine



Key Points

- Epinephrine increases coronary Perfusion pressure and the likelihood of ROSC during CPR
- Epinephrine may decrease microvascular perfusion and prolong post-ROSC hypoperfusion in many organs, including the brain
- On the basis of observational data and limited clinical trials, standard dose epinephrine does not increase and may actually reduce long-term survival and neurological recovery after CPR

The future??

SNP CPR?

- **Sodium Nitroprusside**
- **Potent vasodilator through release of nitric oxide**
- **Clinically used to Tx hypertensive emergencies and CHF**
- **Counter intuitive as after 5-10 mins V-fib cardiac arrest, vasoconstrictors are required to achieve ROSC**
- **Large doses of SNP during CPR do not result in significantly decreased aortic pressure**

Animal Studies of SNP CPR in V-fib Arrest

Definitions:

- eCPR:enhanced CPR= ACDC CPR with ITD and abdominal binding.
- sCPR: standard CPR = CPR as recommended in 2005 AHA Guidelines. Compressions >100/min, Epinephrine every 5 min, Asynchronous positive pressure ventilations @ 8-10/min
- SNP CPR = eCPR + SNP 2 mg at 1 min CPR and 1 mg at 3 min CPR first shock at 6 min CPR

Protocol A: 15 mins. V-fib, then 5 min CPR only, then ACLS intervention epinephrine or SNP

Protocol B: 10 mins. V-fib, then 3 minutes CPR, then shock to PEA, then ACLS as above

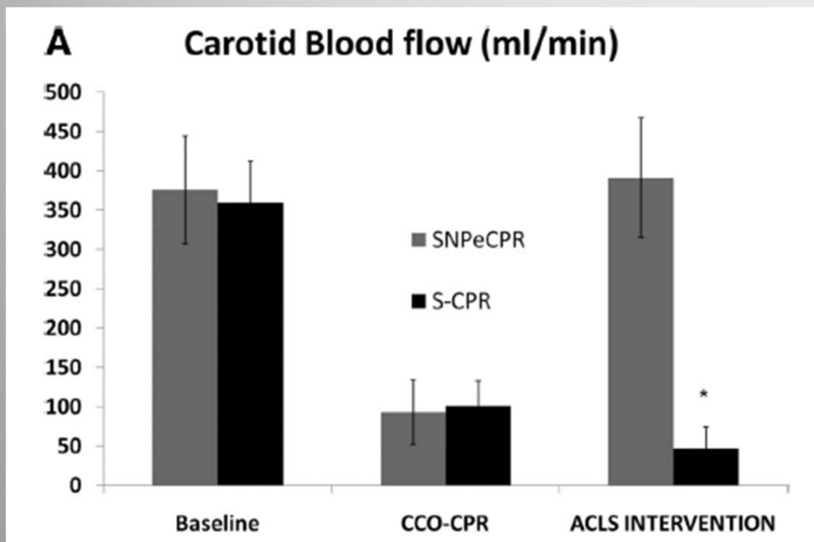
Schultz JC, Segal N, Caldwell E, Sodium nitroprusside-enhanced cardiopulmonary resuscitation improves resuscitation rates after prolonged untreated cardiacarrest in two porcine models. Crit Care Med 2011; 39:2705–2710)

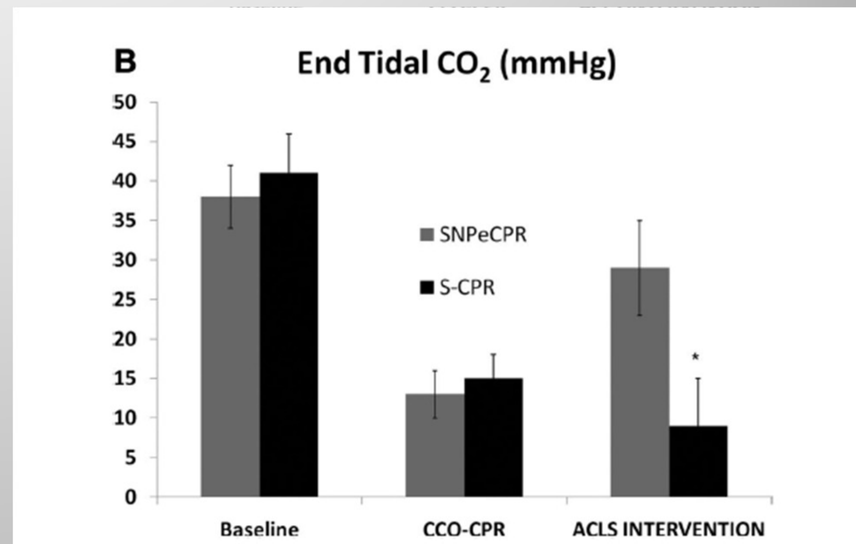
Protocol A

	sCPR	eCPR	SNPeCPR
SBP baseline	107	109	118
SBP during CPR	55	62	67
SBP during ACLS	68	71	95
DBP baseline	78	76	81
DBP during CPR	12	16	29
DBP during ACLS	28	32	48
Coronary Perfusion Pressure			
CPP baseline	76	73	79
CPP during CPR	10	16	23
CPP during ACLS	25	32	41
Carotid Blood Flow			
CBF baseline	389	393	403
CBF during CPR	99	188	427
CBF during ACLS	56	166	489
ROSC:	0/6	0/6	12/12

Schultz JC, Segal N, Caldwell E, Sodium nitroprusside-enhanced cardiopulmonary resuscitation improves resuscitation rates after prolonged untreated cardiacarrest in two porcine models. Crit Care Med 2011; 39:2705–2710)

Protocol B





	S-CPR	SNP e-CPR	p=
ROSC	0/8	7/8	<0.01

Yannopoulos D, Matsuura T, Schultz J, et al. Sodium nitroprusside enhanced cardiopulmonary resuscitation improves survival with good neurological function in a porcine model of prolonged cardiac arrest. Crit Care Med 2011;39: 1269-74

Protocol 24 pigs after 8 mins untreated v-fib randomized into:

- s-CPR (8 pigs) epinephrine 0.5 mg every 5 mins;
- e-CPR (8 pigs) no epinephrine;
- SNPe-CPR (8 pigs) no epinephrine SNP 1 mg every 5 mins.

After 25 minutes of CPR defibrillation was attempted. Animals with ROSC were observed under anesthesia until hemodynamics were stable then surgical repair of vascular access sites and observed for 24 hours. Animals alive at 24 hrs were examined by a veterinarian and given a neurologic score. The following scoring system was used: 1 normal; 2 slightly disabled; 3 severely disabled but conscious; 4 vegetative state; or 5 dead

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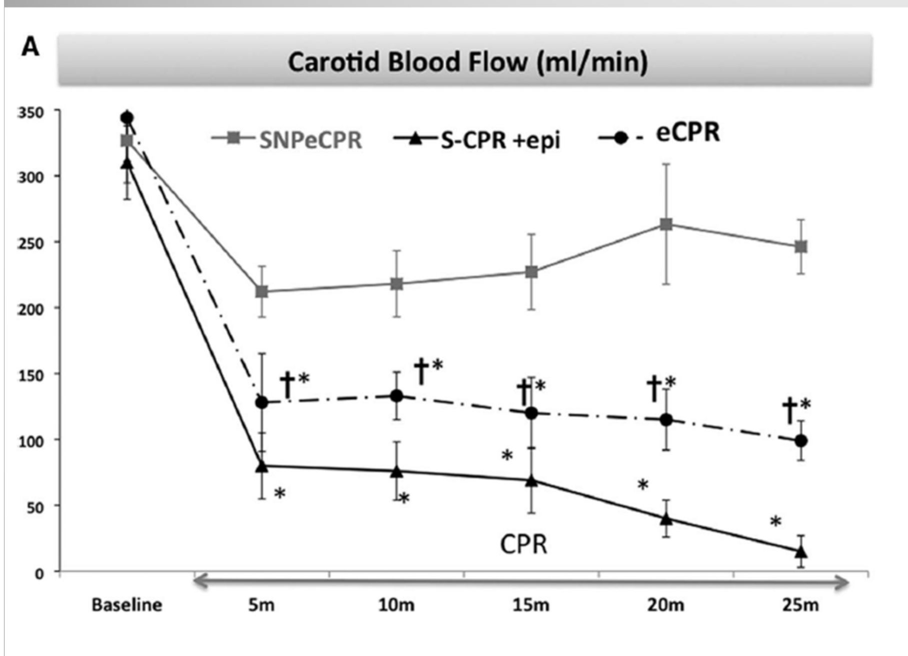
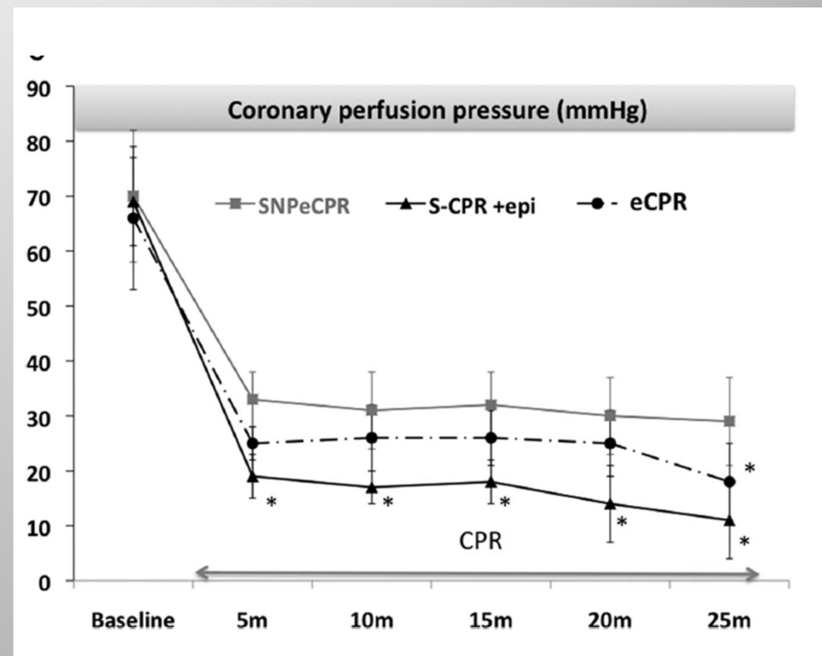
Results:





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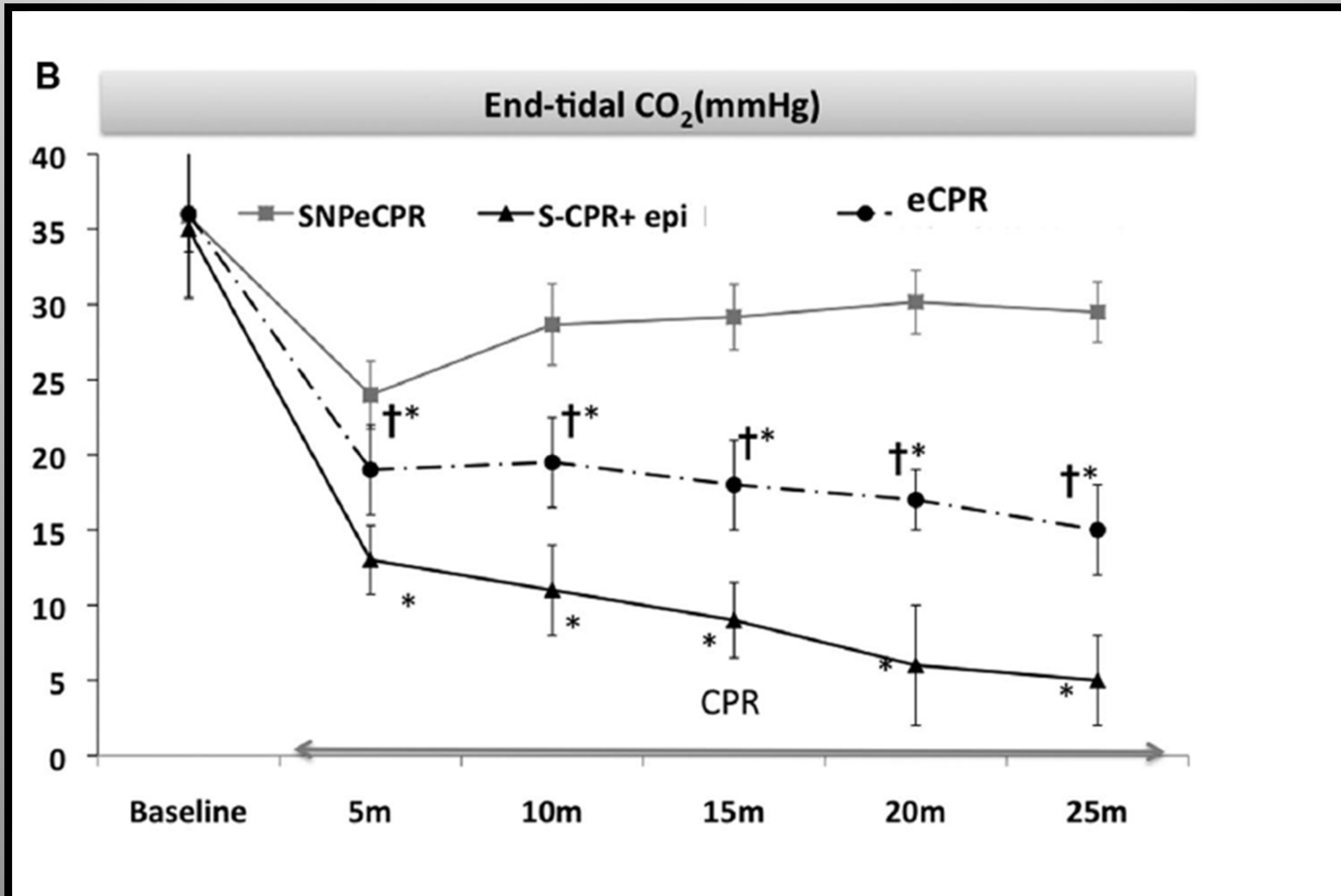

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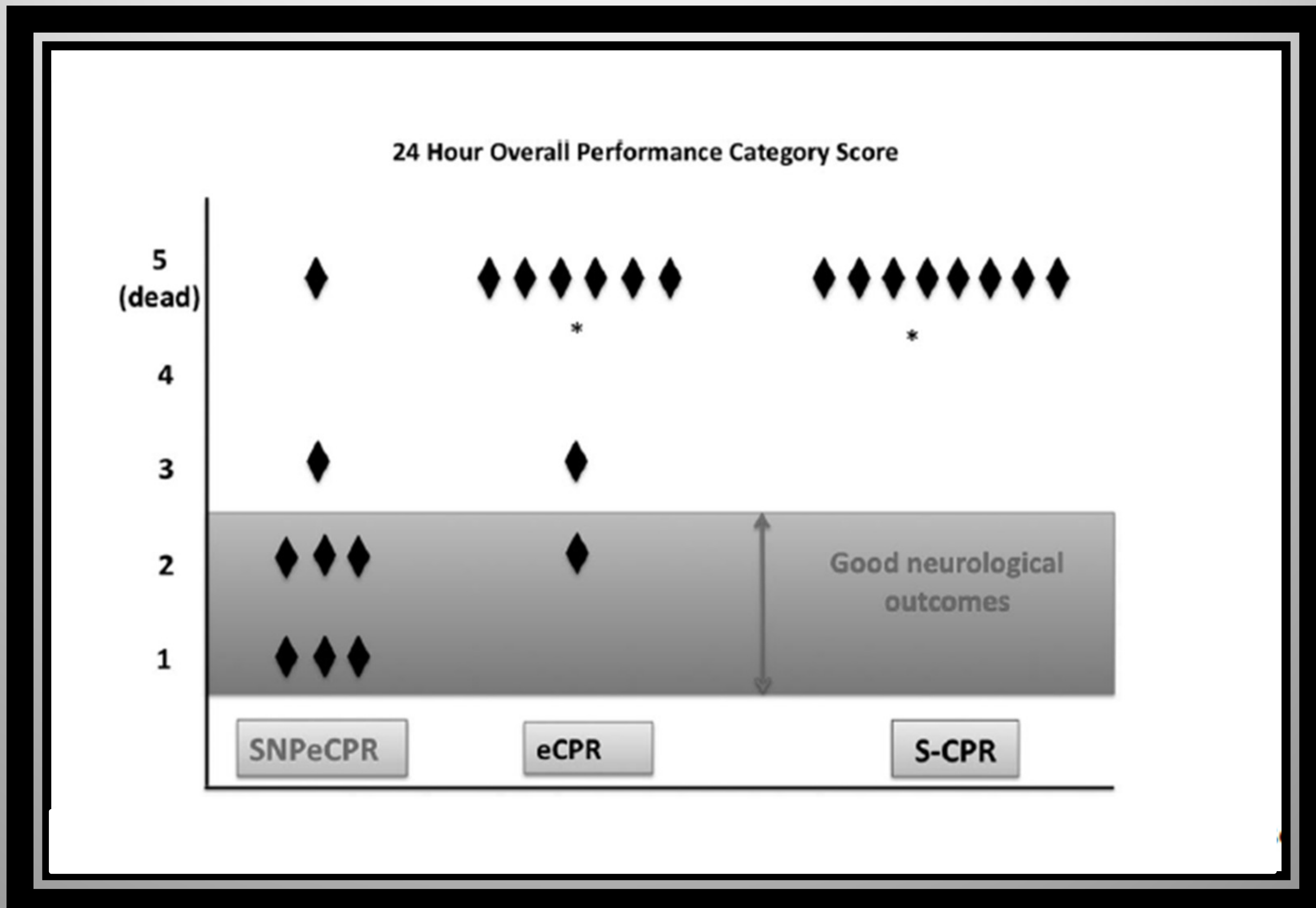

******p* < *.05*** **compared to SNPeCPR**.

Key Points

• SNPeCPR appears to be a promising change in the way we deal with cardiac arrest. Prior pharmacologic approaches to the management of prolonged arrest have focused on the use of vasoconstrictive agents and none have been shown definitely to be beneficial.

• The mechanisms of the beneficial effects of SNP during CPR are likely due to the significant increase in blood flow secondary to nitric oxide, generated by the metabolism of SNP. Exogenous nitric oxide has been shown to alleviate reperfusion injury in a number of organs such as the heart, kidney, and liver. Animal studies have also documented the protective effect of nitric oxide in the early stages of cerebral ischemia and point to the therapeutic potential of SNP in the management of brain ischemic damage

In Summary

• CCR or CCC CPR is beneficial in patients with primary cardiac arrest
• Advanced Airway Management is unnecessary in the initial management of OHCA due to primary cardiac arrest and has been shown to be associated with a worse outcome in a large epidemiological study
• With the exception of ACDC CPR +ITD no CPR adjuncts have demonstrated efficacy in improving outcome from OHCA
• Initiating Therapeutic Hypothermia in the prehospital setting does not improve outcome.
• While active temperature management may be beneficial in comatose survivors of OHCA ideal temperatures have not been defined
• In animal models of OHCA Sodium nitroprusside significantly improves brain and heart blood flow, increases the rate of ROSC and greatly improves neurologic outcome following prolonged V-fib arrest